A Dissertation on

CORRELATION OF FACILITY OF AQUEOUS OUTFLOW AND INTRAOCULAR PRESSURE IN VARIOUS STAGES OF DIABETIC RETINOPATHY – A HOSPITAL BASED STUDY

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CERTIFICATE

This is to certify that the study entitled **"CORRELATION OF FACILITY OF AQUEOUS OUTFLOW AND INTRAOCULAR PRESSURE IN VARIOUS STAGES OF DIABETIC RETINOPATHY – A HOSPITAL BASED STUDY"** is the result of original work carried out by **Dr. K. SATHYA,** under my supervision and guidance at **STANLEY MEDICAL COLLEGE, CHENNAI.** The thesis is submitted by the candidate in partial fulfillment of the requirements for the award of **M.S Degree in Ophthalmology,** course from May 2011 to April 2014 at the Stanley Medical College, Chennai.

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DECLARATION

I hereby declare that this dissertation entitled **"CORRELATION OF FACILITY OF AQUEOUS OUTFLOW AND INTRAOCULAR PRESSURE IN VARIOUS STAGES OF DIABETIC RETINOPATHY – A HOSPITAL BASED STUDY"** is a bonafide and genuine research work carried out by me under the guidance of **Prof. Dr. K . BASKER**, M.S., D.O., HOD, Department of Ophthalmology, Government Stanley Medical College And Hospital , Chennai – 600001.

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ABSTRACT

CORRELATION OF FACILITY OF AQUEOUS OUTFLOW AND INTRAOCULAR PRESSURE IN VARIOUS STAGES OF DIABETIC RETINOPATHY – A HOSPITAL BASED STUDY

Key words: tonography, diabetic retinopathy, aqueous outflow facility.

AIM:

To determine the correlation of aqueous outflow facility and intraocular pressure in various stages of diabetic retinopathy and to determine their relationship with glycemic control of the patient.

MATERIALS AND METHODS:

A total of 150 diabetic patients were included in our study. It included 25 patients with No Diabetic retinopathy, 25 patients each in mild, moderate,severe, very severe Non proliferative Diabetic Retinopathy, 25 patients in Proliferative Diabetic Retinopathy. Patients with bilaterally symmetrical stage of DR were selected for our study. Patients with other systemic complications, closed angle, secondary glaucoma, known case of glaucoma, ocular surgery in the past were excluded.

All patients underwent detailed systemic and ocular examination,applanation tonometry,tonography, perimetry. Systemic examination for glycemic status with FASTING, POST PRANDIAL BLOOD SUGAR, HbA1C.patients with significantly low aqueous outflow facility were followed up every 3 months in this one year study.

RESULTS:

Average aqueous outflow facility was found to be decreased with advance in the stage of diabetic retinopathy in both the eyes. It was found to be statistically significant (RE p-Value=0.000, LE p-Value= 0.000).

Mean intraocular pressure was found to be increased with advance in the stage of diabetic retinopathy in both the eyes. It was found to be statistically significant (RE p-Value= 0.000, LE p-Value = 0.006).

Average aqueous outflow facility was found to be decreased with increase in mean HbA1C level in various stages of diabetic retinopathy in both eyes. (RE r= -0.761, p-Value=0.000, LE r= -0.759, p-Value= 0.000) .

Average IOP was found to be increased with increase in mean HbA1C level in various stages of diabetic retinopathy in both the eyes. It was found to be statistically significant.(REr=0.628, p-Value= 0.000, LE r= 0.608, p-Value= 0.000).

Among the 150 patients 43 patients had significantly reduced aqueous outflow facility at their first visit examination. Hence they were followed up every 3 months.

Among 43 patients advised follow up , 22 patients attended regular follow up of maximum 3 visits every 3 months in this one year study period.

At the end of this one year period 2 of the 22 patients were found to develop early glaucomatous field changes in their visual field. They were also found to have continued poor glycemic control (HbA1C > 6.5), significantly low aqueous outflow facility and intraocular pressure slightly increased compared to the initial recording.

CONCLUSION:

This study has revealed that performing tonography and detecting patients with significantly low aqueous outflow facility can be a guidance for early diagnosis of development of POAG rather than relying on IOP alone .

Hence tonography can be used as one of the examination techniques along with IOP measurement , optic nerve head evaluation and perimetry, for early identification of risk of developing POAG due to reduced aqueous outflow facility in diabetic patients with advanced stages of diabetic retinopathy.

INTRODUCTION

Diabetic retinopathy and glaucoma are among the leading causes of visual impairment. If not diagnosed and treated earlier they can lead to irreversible visual impairment and blindness. Various methods are under evaluation for early diagnosis and treatment of these conditions.

Glaucoma is a chronic progressive optic neuropathy caused by a group of ocular conditions which lead to damage to optic nerve with loss of visual function. Increase in intraocular pressure is the most commonly known risk factor.

Intraocular pressure is determined by the balance between aqueous production and aqueous outflow. Imbalance of the two can lead to increased intraocular pressure, which is one of the reversible risk factors for glaucoma. Aqueous outflow is one of the important

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PART – I

INTRODUCTION

Diabetic retinopathy and glaucoma are among the leading causes of visual impairment. If not diagnosed and treated earlier they can lead to irreversible visual impairment and blindness. Various methods are under evaluation for early diagnosis and treatment of these conditions.

Glaucoma is a chronic progressive optic neuropathy caused by a group of ocular conditions which lead to damage to optic nerve with loss of visual function. Increase in intraocular pressure is the most commonly known risk factor[1].

Intraocular pressure is determined by the balance between aqueous production and aqueous outflow. Imbalance of the two can lead to increased intraocular pressure, which is one of the reversible risk factors for glaucoma. Aqueous outflow is one of the important determinants of intraocular pressure.

Quantification of aqueous outflow facility brings us close to the basic mechanism of abnormal aqueous humor dynamics in glaucoma than measurement of intraocular pressure alone[2]. Intraocular pressure reflects the combined effect of three factors: aqueous production ,

aqueous outflow and episcleral venous pressure, only one of which is known to be abnormal in most types of glaucoma.

Diabetes Mellitus is one type of metabolic disorder[7]. It is characterised by chronic hyperglycemia which leads to long term damage and dysfunction of various organs like eyes, kidney, nerves, heart, blood vessels,etc.. In India about 35 million people with diabetes mellitus are present till date.

Diabetes mellitus being an important ocular risk factor, besides causing diabetic retinopathy and other ocular manifestations also affects the intraocular pressure by affecting the outflow facility[6].

When the cause for an abnormal pressure is under issue, **tonography** remains a very informative and surprisingly convenient procedure for determination of outflow facility. Tonography can be done as an outpatient procedure using non- invasive methods.

Since aqueous outflow is one of the important determinants of intraocular pressure – the present study is undertaken to correlate the aqueous outflow facility with the diabetic patients at various stages of

diabetic retinopathy[24], also taking into account their glycemic control. This study was done among patients treated at Stanley medical college and hospital, at Chennai.

REVIEW OF LITERATURE

The French Dr Antoine- demours (1818) made the first excellent description of glaucoma with raised intraocular pressure.

Dr William McKenzie (1835) mentioned the essential feature of raised eye tension. He was a Scottish clinician .

In 1873 Leber discovered that, aqueous humor is the result of an ongoing dynamic process involving the eye and the circulatory system[5].

Grant introduced tonography in 1950[2,23].

An electronic schiotz tonometer that enhanced precision of pressure induced flow was discovered by V. Mueller[3] .

The definition of glaucoma as a disease of optic nerve (glaucomatous optic neuropathy) caused by many factors which were called as risk factors was described by Dr. drance (1973) .

The name Schlemm's canal is given after Friedrich Schlemm (1795–1858), a German anatomist[5].

Aretaeus belongs to Cappadocia. He was an eminent physician in a Pneumatic school. He introduced the term diabetes (the Greek verb

diabaino = pass through) to define the condition in which a large quantity of urine passes through kidney in 2nd century AD .

Friederick Banting and Charles Best in 1921 extracted insulin from the pancreas.

In 1856 - Diabetic macular changes was reported first by Eduard Jaeger .

In 1876, Wilhelm Manz - reported proliferative changes in diabetic retinopathy and the importance of tractional retinal detachments and vitreous haemorrhages.

Arthur James Ballantyne - diabetic retinopathy represents a unique vasculopathy.

Beaver Dam Study in 1994 analysed the relationship between open angle glaucoma and late onset diabetes.

Los angeles Latino Eye Study in 2008 concluded that presence of Type2DM was associated with increased risk of having open angle glaucoma.

Matsuoka et al, in 2012 reported that there is significantly higher IOP in diabetic patients and positive correlation of IOP with HbA1c levels in patients with diabetic retinopathy.

AQUEOUS OUTFLOW PATHWAY ANATOMY

Aqueous outflow pathway is located at the angle of the anterior chamber[3].

It has the following parts:

1. **Trabecular Meshwork**
    - Uveal Meshwork
    - Corneoscleral Meshwork
    - Juxta Canalicular Meshwork
2. **Schlemm's Canal**
3. **Collector Channels**
4. **Episcleral Veins**

1. Trabecular Meshwork:

It is a sieve like structure. It bridges the scleral sulcus, and converts it into a tube and accomodates schlemm's canal[44]. It consists of Hyaluronic acid (GAG) & Chondroitin, Heparin, dermatan and keratan

sulphates . It also consists of Fibronectin, elastin, laminin, collagen (I, III, IV, V, VI), myosin.

- Uveal meshwork:

It is the innermost part. It extends from iris root and ciliary body to schwalbe's line. It is cord like and 2-3 layers thick. The arrangement of it's trabeculae creates irregular openings 25-75 µm size. **Trabeculae have 3 concentric layers**

1. Central collagenous core
2. Middle basement membrane
3. Enclosing trabecular cell

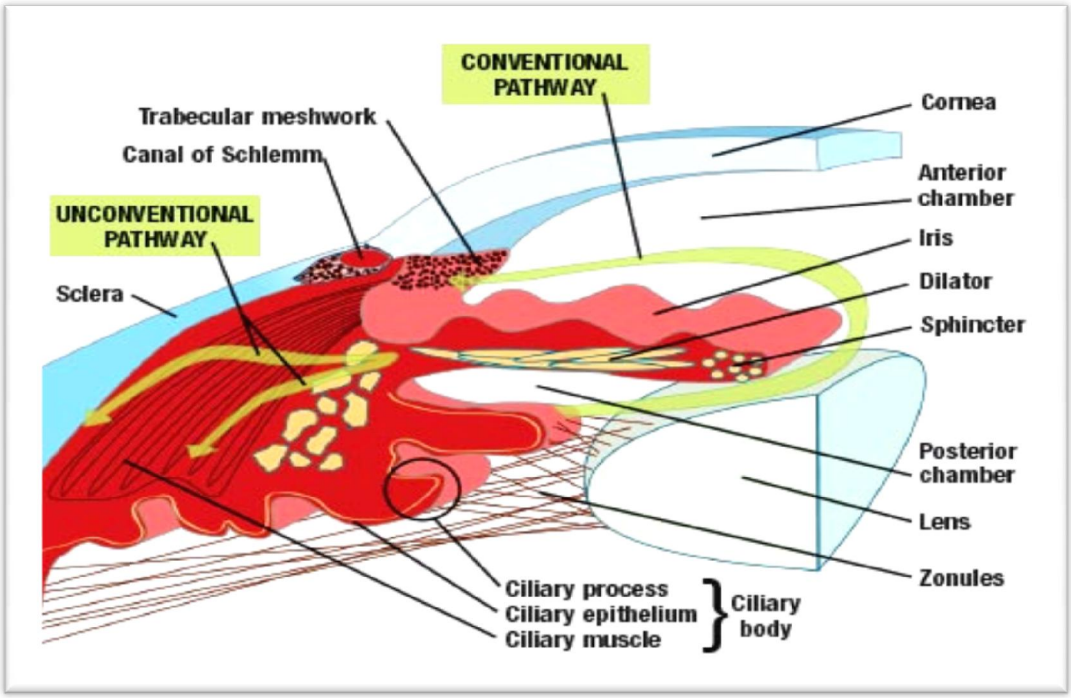
- Corneoscleral Meshwork:

It is the middle portion. It extends from the scleral spur to the lateral wall of the scleral sulcus . It consists of flat sheets of trabeculae with elliptical openings (5-50µ). Openings become smaller as the trabecular sheets approach schlemm's canal (1-2µ).

➢ Juxtacanalicular Meshwork:

It is the outermost portion ,**offers the normal resistance to aqueous outflow**[2]. Layers of connective tissue (2-5 layers) lined on either side by endothelium are present . This narrow part (2-20µ) connects corneo-scleral meshwork with schlemm's canal.

AQUEOUS OUTFLOW PATHWAY



Schlemm's Canal[28]:

It is an oval channel lined by endothelium present circumferentially in the scleral sulcus. Its inner wall cells are spindle shaped and irregular. It contains giant vacuoles. Outerwall cells are smooth and flat. Numerous openings on the outer wall form the collector channel. Torus or lip like thickening and septa are present to keep the canal open.

2. Collector Channels:

These are intra scleral aqueous vessels. They are 25-35 in number, leave at oblique angles and end in episcleral veins.They are lined by vascular endothelium. They have no valves.

These intrascleral aqueous vessels can be divided into two systems.

a. Direct system (8 Large Vessels):

It has short Intrascleral course and terminates in episcleral veins. On slit lamp examination it appears as clear vessels with aqueous called **Aqueous veins by Ascher**. They are also called as **laminated veins of Goldmann.**

b. Indirect system :

Numerous fine channels drain into

- Deep intrascleral plexus
- Mid intrascleral plexus
- Episcleral venous plexus

Ultimately they end in episcleral veins.

3. Episcleral Veins:

They drain into Anterior Ciliary Vein → Superior Ophthalmic Vein → Cavernous Vein.

AQUEOUS HUMOUR DYNAMICS

Aqueous humor dynamics includes aqueous inflow (ie. Formation) and outflow (ie. Drainage)[2]. Aqueous humor flows from posterior to anterior chamber through pupil against slight physiological resistance.

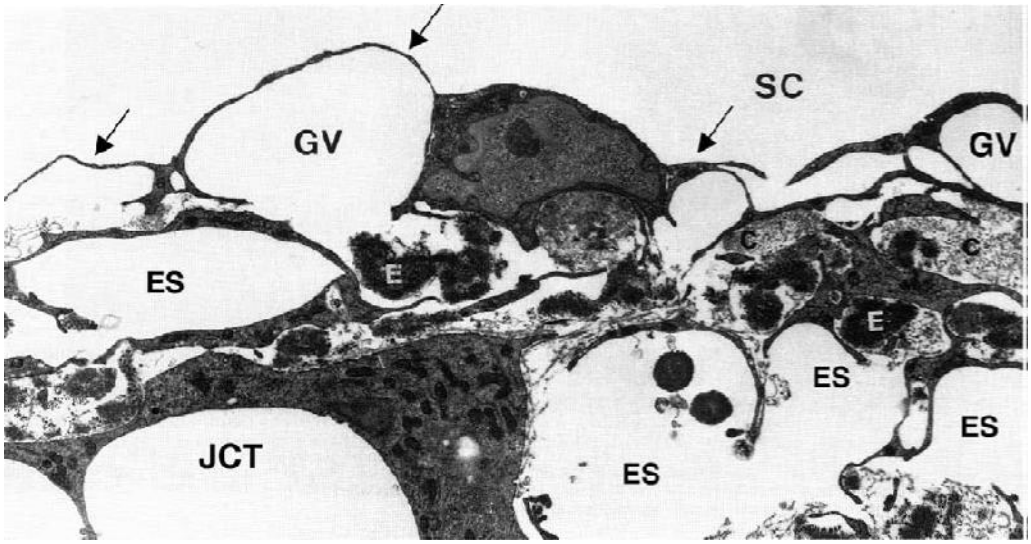
In the anterior chamber there is convection (thermal) current due to temperature gradient between anterior and posterior part of the anterior chamber.

The aqueous humor in the posterior part of anterior chamber moves up along warm iris and in the anterior part moves down along the cooler cornea.

From the anterior chamber , aqueous is drained out by two routes:

- Trabecular (conventional) outflow
- Uveoscleral (unconventional) outflow

Transmission electron micrograph of the inner wall of Schlemm's canal (SC) of a human eye:



Trabecular outflow:

It is the main outlet for aqueous from anterior chamber. About 90% of the aqueous is drained out via this route. Free flow occurs from trabecular meshwork upto inner wall of schlemm's canal which provides least resistance[45].

Mechanism of transport across inner wall of schlemm's canal:

Vacuolation theory is the most accepted view. According to it transcellular spaces exist in the endothelial cells forming inner wall of schlemm's canal. These open as a system of vacuoles[46] and pores ,

primarily in response to pressure , and transport the aqueous from the juxtacanalicular connective tissue to schlemm's canal.

From schlemm's canal[48] the aqueous is transported via 25-35 external collector channels into the episcleral veins by direct and indirect systems. A pressure gradient between intraocular pressure and intrascleral venous pressure (about 10 mm hg) is responsible for unidirectional flow of aqueous.

Uveoscleral outflow:

It is responsible for about 10% of the total aqueous outflow. Aqueous passes across the ciliary body into the suprachoroidal space and is drained by the venous circulation in the ciliary body , choroid and sclera.

GLAUCOMA

Glaucoma is a group of disorders characterised by progressive optic neuropathy resulting in characteristic appearance of the optic disc and a specific pattern of irreversible visual field defects that are associated frequently but not invariably with raised intraocular pressure. Thus intraocular pressure is the most common but not the only risk factor for the development of glaucoma.

Glaucoma in greek means **clouded or blue-green hue.**

Classification:

Glaucoma may be classified as follows:

1. **Congenital and developmental glaucomas:**
   - Primary congenital glaucoma(with no associated anomalies)
   - Developmental glaucoma(associated with anomalies)
2. **Primary adult glaucomas:**
   - Primary open angle glaucoma
   - Primary angle closure glaucoma
   - Primary mixed mechanism glaucoma

3. Secondary glaucomas.

Glaucomatous ocular damage can be due to the following pathogenesis:

As per the definition of glaucoma all glaucomas are characterised by progressive optic neuropathy. Progressive optic neuropathy results from death of retinal ganglion cells in a typical pattern which results in characteristic optic disc appearance and visual field defects.

Pathological mechanism of retinal ganglion cell death[3]:

It occurs due to pathological block in transport of growth factors(neurotropins) from the brain to the retinal ganglion cells. This initiates a damaging cascade , and the cell is unable to maintain its normal function. They undergo apoptosis and also trigger apoptosis of adjacent cells.

Retinal ganglion cell death is associated with loss of retinal nerve fibres. As this extends beyond the normal physiological overlap of functional zones the characteristic optic disc changes and specific visual field defects become apparent overtime.

Etiological factors:

Factors involved in retinal ganglion cell death and hence glaucomatous optic neuropathy are:

A. Primary insults:

1) Raised intraocular pressure (Mechanical Theory): It causes mechanical stretch on lamina cribrosa → axonal deformation and ischemia by altering capillary blood flow. So neurotrophins are not able to reach in sufficient amount to retinal ganglion cells for their survival.

2) Pressure independent factors (Vascular Insufficiency Theory): Factors affecting vascular perfusion of optic nerve head in the absence of raised intraocular pressure are also implicated in glaucomatous optic neuropathy[14]. These factors include-

    - Defective autoregulatory mechanism of blood flow
    - Vasospasm
    - Systemic hypotension [10]– especially nocturnal dips in patients on anti- hypertensive medications.

- Other factors- acute blood loss, abnormal coagulability profile[9].

B. Secondary insults (excitotoxicity theory):

Degeneration of neurons is also driven by certain toxic factors. They are glutamate(excitatory toxin), oxygen free radicals, or nitric oxide. They are released when retinal ganglion cells undergo death due to primary insults.

Optic nerve head changes in glaucoma[3]:

There is no first sign of glaucomatous nerve damage nor there is a single pattern of damage in optic disc. Rather there are many patterns by which the nerve becomes damaged.

1. Features related to loss of neuroretinal rim tissue: notching, pseudopit, diffused rim loss.

2. Features related to development, enlargement or alteration of the cup: asymmetry of cup size, overpass cupping, vertical extension, concentric enlargement of the cup, temporal unfolding, nasal cupping, C:D ratio increase, laminar dot sign.

3. Features related to change of color of optic disc tissue: pallor and cupping.

4. Features related to change of blood vessels of the disc: optic disc haemorrhage, nasalization of vessels, bayonetting of vessels, baring of circumlinear vessels .

5. Peripapillary area Beta zone atrophy.

OPTIC NERVE HEAD CHANGES

Advanced glaucomatous cupping

Localized rim loss inferiorly

GRADING OF ANGLE DEPTH

There are three standard grading system for anterior chamber angle depth.

1. Scheie grading system
2. Shaffer grading system
3. Spaeth grading system

SCHEIE GRADING SYSTEM:

Grade	Parts visible
1	Angle is widely open
2	Angle permits visibility to scleral spur
3	Angle permits visibility to trabecular meshwork only
4	Angle is appositionally closed to Schwalbe's line

SHAFFER GRADING SYSTEM[4]:

Grade	Angle width in degrees	configuration	Structures visible
4	40	Wide open	SL, TM, SS, CBB
3	30	Open angle	SL, TM, SS
2	20	Moderately narrow	SL, TM
1	10	Very narrow	SL only
S	< 10	Slit angle	No angle structures seen
0	0	Closed	No angle structures seen

SL – schwalbe's line, TM – Trabecular meshwork,

SS – Scleral spur , CBB – Ciliary body band

SPAETH GRADING SYSTEM OF ANTERIOR CHAMBER ANGLE

Site of iris insertion:

Anterior to schwalbe's line = A

Behind Schwalbe's line = B

At scleral spur = c

Normal deep angle recess = D

Extremely deep angle recess = E

Angle width in degrees:

10, 20 , 30, 40, 50

Peripheral iris curvature:

Concave = q

Flat = r

Steep (convex) = s

PERIMETRY AND VISUAL FIELDS IN GLAUCOMA

Perimetry is the procedure for determining the extent of the visual fields[4].

It can be classified as follows:

- Kinetic and static perimetry:
- Peripheral and central field charting:
- Manual and automated perimetry:

1. **Kinetic perimetry:** Here a stimulus is moved from a peripheral non-seeing point towards the centre till it is perceived to establish the isopters. Eg: Confrontation method, Lister's perimetry, Tangent screen scotometry, Goldmann perimetry.

2. **Static perimetry:** Here a stimulus is presented at a predetermined position for a preset time with varying luminance in the field of vision. Eg: Goldmann perimetry, Friedman perimetry, Automated perimetry.

Automated perimetry:

It is computer assisted and tests visual fields by a static method.

They automatically test suprathreshold and threshold stimuli and quantify depth of visual field defect.

Commonly used automated perimeters are – Octopus, Field Master, Humphrey Field Analyser.

VISUAL FIELD LOSS IN GLAUCOMA:

Generalised loss:

Diffused loss of sensitivity.

Increased variability.

Hemifield asymmetry.

Localized loss:

Early defects: paracentral scotoma,nasal step, temporal wedge defect.

Late defects: arcuate scotoma, annular scotoma, altitudinal defect.

Advanced field loss:

Retained central vision and/or temporal island.

Split of fixation.

Loss of central island and or temporal field.

AQUEOUS OUTFLOW PATHWAY CHANGES IN DIABETES MELLITUS

Individuals with diabetes are found to have high frequencies of glaucoma and increased intraocular pressure[11]. It has been ascribed that there is increased accumulation of extracellular matrix components like fibronectin and glycosaminoglycans in the aqueous outflow pathway.

The trabecular meshwork in the chamber angle is found to function as a self-cleaning filter and to participate in the regulation of aqueous outflow and control of intraocular pressure.

Connective tissue in the trabecular meshwork beam contains extracellular matrix proteins like fibronectin, laminin, heparin sulfate, and collagen types 1,3,4,5,6.

Davis et al[42] have reported that the glucose levels in aqueous humor of patients with diabetes was significantly higher. **Trabecular meshwork cells undergo biochemical alterations** when they are exposed to such amount of high concentration of glucose in aqueous humor .

There occurs fibronectin over expression at both mRNA and protein level in trabecular meshwork[18]. The high glucose level also

reduces trabecular meshwork cell proliferation as in vascular endothelial cells. The chemoattractant potential of fibronectin in aqueous humor causes decrease in trabecular meshwork cellularity[47].

The regulation of aqueous outflow in the trabecular meshwork is impaired by these changes in diabetes[16] ,leading to reduced aqueous outflow facility and hence increased intraocular pressure.

Fibronectin overexpression is induced by high glucose . This may be a common biochemical link contributing to two main changes:

1. Thick vascular basement membrane in diabetic microangiopathy
2. Altered structural content, resiliency, reduced cellularity and hence blockage of aqueous outflow in trabecular meshwork.

Elevated IOP in diabetes could be explained in part by higher insulin resistance and chronic hyperglycemia . In the eye insulin acts as a vasodilator through nitric oxide. Insulin and IOP can have some common effect.

Nitric oxide decreases IOP acutely by increasing trabecular outflow facility . IOP falls acutely in response to nitric oxide induced contraction of cell volume. The mediators for this process are soluble guanylate cyclase, PK G, and Ca2+ -activated K+ channel[21] . In fact,

these regulatory elements may associate insulin with changes in outflow facility. Reduction in insulin and hence nitric oxide decreases this outflow facility.

Further there is reduced antioxidant[51] in diabetes at the level of trabecular meshwork as in all other parts of the body . Hence there is early trabecular damage leading to increased outflow resistance[49].

Due to the **accumulation of advanced glycation end products** (AGE) there will be **Oxidative stress[19]**. Advanced glycation end products (AGE) are nonenzymatic protein cross-links. They may interfere with the structural properties of the protein. This leads to some deleterious effects.

In the meshwork, tissue transglutaminase is present and due to its advanced glycation it causes increased TGF 1,2 which causes polymerization of fibronectin leading to irreversibly cross-linked extracellular matrix proteins. The pathological changes due to AGE products in the outflow pathways and role of AGE in glaucoma is being still under research.

Several molecules[21] (TGFβ2, VEGF, endothelin, PAI, and soluble CD44) are elevated in the aqueous humor of POAG when compared to normal aqueous humor. These molecules may influence

trabecular cells to change their normal phenotype ,ie. trabecular cells could respond to elevated aqueous humor levels of TGF-β2 by secreting additional extracellular matrix molecules.

TONOMETRY

It is an equipment used for measurement of intraocular pressure by utilizing physical natures of the eye ,without the need to cannulate the eye[1] .

The physical characters of the normal cornea determine the limits of accuracy of tonometry

CLASSIFICATION :

1. **APPLANATION TONOMETRY**[4]
   - Goldmann Applanation Tonometry
   - Perkin's Applanation Tonometry
   - Non-Contact Tonometry
2. **INDENTATION TONOMETRY**
   - Schiotz Tonometer
   - Pneumotonometer
   - Tono-Pen
3. **REBOUND TONOMETRY**

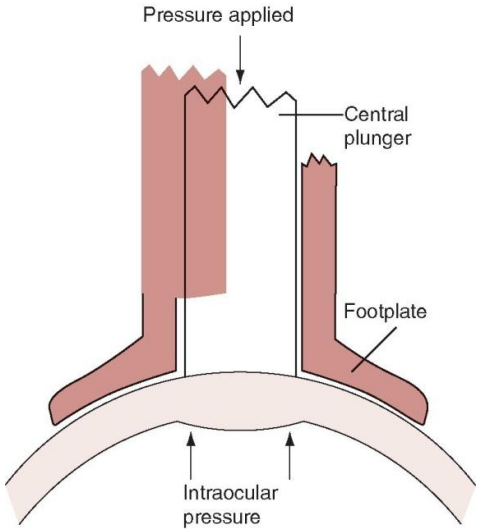
Schiotz tonometry:

With the Schiotz tonometer ,various standard weights are applied to the cornea via a plunger. Cornea is indented by a plunger and the deformation of the globe is recorded from a scale. The IOP is then calculated from these two values.

The plunger moves in the center of the instrument in a vertical plane and passes through a curved footplate that sits on top of the cornea with the patient in the supine position.

SCHIOTZ TONOMETER

PRINCIPLE OF SCHIOTZ TONOMETER



A "holder" fixes the footplate on the cornea .But it allows the plunger and attached weights to move in the vertical direction freely.

On placing the tonometer on the eye, there occurs distention of the globe due to corneal indentation. This corneal indentation and globe distention both are indicated by the scale reading.

Friedenwald's formula relates this distention to the IOP. The formula requires a constant "K" or the "coefficient of ocular rigidity,".

Ocular rigidity:

Ocular or sclera rigidity is an expression of strechability of the eye in response to increase in intraocular pressure.

The value of coefficient of scleral rigidity (K) for an individual eye was determined by **Friedenwald**[2]. It was calculated from two tonometric scale readings using different weights. **Then Friedenwald's "nomogram**" allows one to graphically calculate K. The average normal value was 0.0215 as calculated by him.

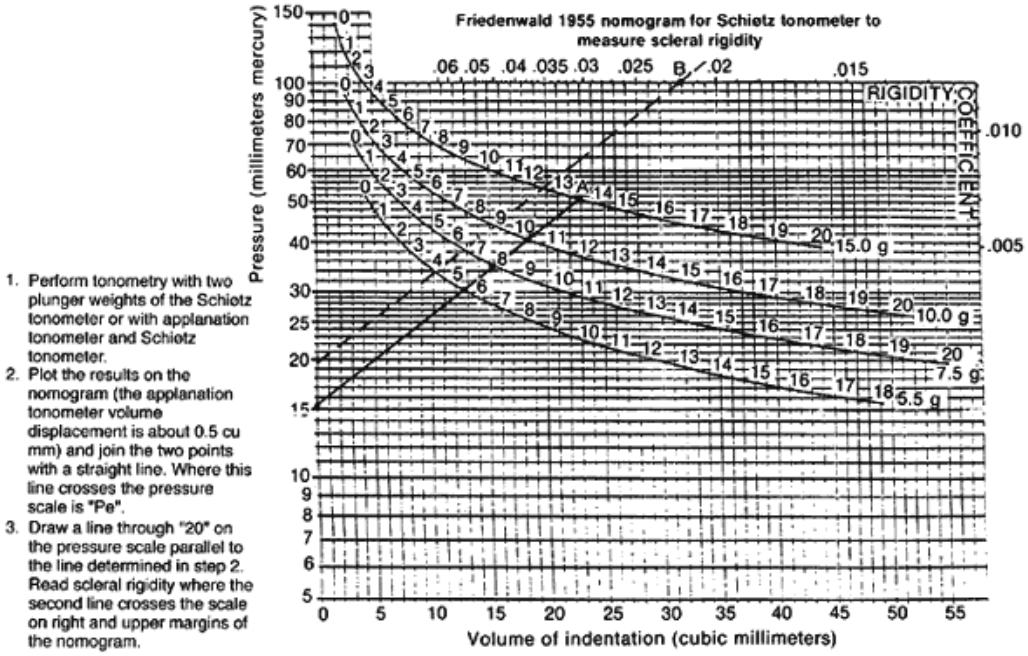
At present , there are simplified tables which gives the K and IOP values from the paired scale readings on the involved eye .

Limitations :

If the true K of the eye is higher than the average K, true IOP will be overestimated.

A false-low IOP will occur if the true K is less than the average K.

Friedenwald's "nomogram"

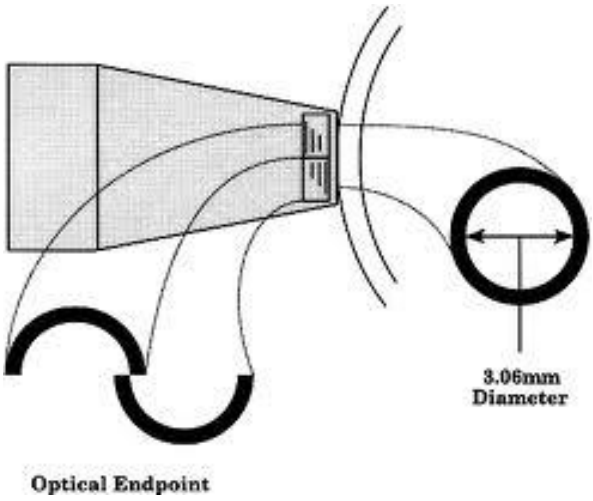


1. Perform tonometry with two plunger weights of the Schiotz tonometer or with applanation tonometer and Schiotz tonometer.
2. Plot the results on the nomogram (the applanation tonometer volume displacement is about 0.5 cu mm) and join the two points with a straight line. Where this line crosses the pressure scale is "Pe".
3. Draw a line through "20" on the pressure scale parallel to the line determined in step 2. Read scleral rigidity where the second line crosses the scale on right and upper margins of the nomogram.

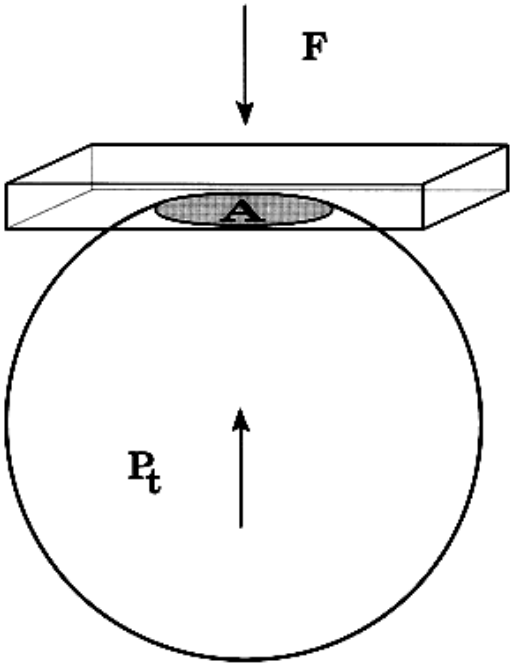
High ocular rigidity has been reported in :

- high hyperopia,
- chronic glaucoma,
- vasoconstrictor therapy.

APPLANATION TONOMETER



IMBERT FICK'S LAW



- **Goldmann applanator:**

Applanation tonometry is based on the **Imbert-Fick's principle.** It states that for an ideal sphere the pressure (P) inside the sphere is equal to the force (F) required to applanate (flatten) its surface, divided by the area (A) of flattening:

$$P = F/A \quad \text{or} \quad F = PA.$$

The ideal sphere will be thin-walled, dry and flexible. The cornea has none of these three. Hence there are two other significant forces at work.

The force of capillary attraction (T) between the tonometer head and the tear film is additive to the external force. In addition, a force

(C), is needed to flatten the cornea which is comparatively inflexible. Hence,

$F = PA$, becomes

$F + T = PA + C$, or

$P = (F + T - C) / A$

The Goldmann applanator is designed so that A will be 7.35 mm 2.

To achieve this, the diameter of flattening of the cornea should be 3.06 mm .

With this value for A, the opposing forces of capillary attraction and corneal inflexibility cancel out. In addition, with this value for A the IOP in millimeters of mercury (mmHg) is equal to ten times the force applied to the cornea which is given in grams.

The tonometer "tip," a tapered plastic cylinder containing a biprism, is the contact point with the cornea. The tip is connected by a rod to the body of the tonometer which contains an adjustable spring that provides the appropriate applanating force. The force is altered manually with a knob that contains a scale indicating the force applied

in grams. At the end-point, the reading in grams is multiplied by 10 to convert to millimeters of mercury.

The biprism splits the image of the circle of contact into two semicircles. When these semicircles just touch at their inner margin, a 3.06-mm diameter circle of cornea is applanated.

Precautions:

- Valsalva maneuvers, or breath holding should be avoided.
- The semicircles should be clear with distinct margins.
- Wider, blurred semicircles result in false-high readings as does vertical misalignment.
- Under estimation of the true IOP will occur if fluorescein is not used.
- False pressure readings occur in corneal astigmatism.

OTHER TONOMETERS:

Perkins applanation tonometer

Draeger applanation tonometer

Mackay- Marg tonometer[3]

Tono – pen tonometer

Pneumotonometer

Noncontact Air- puff tonometer

Dynamic contour tonometer

Ocular response analyser

TONOGRAPHY

Tonography is a dynamic test of the ability of the eye to recover from the elevation of intraocular pressure induced by schiotz tonometry.

Such recovery occurs primarily through an increased rate of aqueous humor flow out of the eye. Hence the test is a measure of facility of aqueous humor outflow[2]. In glaucoma there is reduced aqueous outflow facility as there is increased resistance to aqueous outflow.

Thus quantification of aqueous outflow facility brings us close to the basic mechanism of abnormal aqueous dynamics in glaucoma than measurement of intraocular pressure alone.

Methodology:

Tonography was introduced by Grant in the year 1950[23].

Aqueous humor outflow is measured by various techniques each with its own limitations and advantages.

TRABECULAR OUTFLOW FACILITY:

Some resistance to the aqueous outflow is given by juxtacanalicular part of trabecular meshwork. This resistance is needed so that a steady-state of IOP is maintained normally. The trabecular meshwork's compliance can be measured with this trabecular outflow facility[28].

Techniques:

1) **Non -Invasive techniques**

   a) Tonography - with schiotz tonometer,

   b) Tonography with pneumatic tonometer.

   c) Fluorophotometry[25] .

2) **Invasive techniques-**

   a) constant pressure perfusion technique,

   b) flow-to-blood method with radioisotope infused in the anterior chamber.

Tonography - with schiotz tonometer:

Here, under topical anaesthesia Probe of schiotz tonometer's is placed on the cornea of the subject for 4 minutes , patient being in supine position .The IOP initially rises due to the weight placed on the cornea, but, in course of time, the pressure gradually decreases. This is due to the following reason: Aqueous humor will drain at an increased rate via the drainage pathways from the anterior chamber . The decrease in IOP during the procedure is thought to be mainly due to increased drainage of aqueous humor via the trabecular meshwork from the anterior chamber.

The outflow facility is determined from reference tables using initial reading and reading of schiotz at the end of 4 minutes (Annexure). While testing if there is little decrease in IOP , then the fluid flow rate would be small as in reference tables[36], and hence there will be less trabecular outflow facility. In ocular hypertension with or without glaucoma this finding usually occurs .

Disadvantage: varies with change in ocular rigidity (pneumatic tonometer is less affected by it).

Fluorophotometry :

Here by measuring the rate of disappearance of a tracer from the anterior chamber Aqueous flow (F) is determined[26] . Then IOP and aqueous flow are reduced by medication . The drug-induced change in IOP (IOP2 - IOP1) is then measured with tonometry. The change in aqueousflow (F2 - F1) is then measured by fluorophotometry.

Outflow facility is calculated by Equation :

$$C = (F2 - F1)/(IOP2 - IOP1).$$

Invasive Methods:

The two-level, constant-pressure perfusion technique is an invasive procedure that is used to measure outflow facility in research animals[27].

These are mostly used for enucleated eyes of humans. But in clinical studies they cannot be used .

The flow-to-blood method is the most reliable technique from which we can measure trabecular outflow facility. After infusing a radioactive isotope into the anterior chamber at a set pressure (IOP1) & time , the rate of its accumulation in the blood is assumed to be

trabecular outflow (F1). Then at a different level of pressure (IOP2) infusion of isotope is done , and the new rate of it's accumulation in the blood is assumed to be a new trabecular outflow (F2). Then outflow facility determined as for fluorophotometry.

UVEOSCLERAL OUTFLOW FACILITY :

Overview:

Uveoscleral outflow is another drainage route for aqueous humor from the anterior chamber. Here it leaves into the ciliary muscle. Here it diffuses out of the eye in many directions . The route of this outflow is poorly defined. Also it is a pressure independent outflow pathway .

Measurement techniques for Uveoscleral Outflow :

Non Invasive Method :

The noninvasive means to assess uveoscleral outflow (Fu) is by calculation using the following equation :

$$Fu = F - C(IOP-Pv)$$

Fluorophotometry measures Aqueous humor flow (F). IOP is measured by tonometry. Episcleral venous pressure (Pv) is measured by venomanometry.

Invasive methods:

Compared to the mathematical calculation these procedures are more direct. But in clinical studies they cannot be used .

Intracameral tracer method:

A radioactive or fluorescent tracer is infused into the anterior chamber for a specific period of time at a predetermined pressure. Uveoscleral outflow is determined from the total amount of tracer found in the uvea and sclera during the specified interval of time.

Indirect isotope method:

In this procedure a radioactive tracer is infused into the anterior chamber. Then the rate by which of the tracer's appear in the blood (trabecular outflow) and tracer's disappear from the anterior chamber (aqueous flow) is monitored . The difference between aqueous flow and trabecular outflow is Uv.

COMPLICATIONS IN EYE DUE TO DIABETES MELLITUS[7,29]

1. Diabetic retinopathy[13]
2. Cataract[29]
3. Anterior ischemic optic neuropathy
4. Diabetic papillopathy
5. Ocular movement disorders
6. Glaucoma[6,12] – primary open angle glaucoma , neovascular glaucoma
7. Retinal vein occlusion[7]
8. Retinal artery occlusion
9. Corneal diseases

DIABETIC RETINOPATHY

Diabetic retinopathy refers to retinal changes seen in patients with diabetes mellitus.

It's incidence has increased due to increase in life expectancy of patients with diabetes. It is becoming one of the leading causes of blindness.

Etiopathogenesis:

Risk factors for diabetic retinopathy are:

- Duration of diabetes
- Sex
- Poor metabolic control
- Heredity
- Pregnancy
- Hypertension
- Anemia
- Nephropathy

- ❖ Others- smoking, obesity, hyperlipidemia.

Pathogenesis:

It is a microangiopathy affecting retinal precapillary arterioles, capillaries and venules.

Capillaropathy:

I. Degeneration and loss of pericytes

II. Proliferation of endothelial cells

III. Thickening of basement membrane and occlusion

Hematological changes:

I. Deformation of erythrocytes and rouleaux formation

II. Changes in RBC and hence defective oxygen transport

III. Increased plasma viscocity

IV. Increased stickiness and aggregation of platelets

These changes result in microvascular occlusion and leakage.

Retinal vascular changes in diabetic retinopathy[15]:

1) Capillaries:

   Occlusion, dilation,

   microaneurysms,abnormal permeability.

2) Arterioles:

   Narrowing of terminal arterioles,

   Occlusion ,Sheathing.

3) Veins:

   Tortuosity, Looping ,

   Beading , Sausage like segmentation.

Classification :

There are various types of classifications of Diabetic Retinopathy.

It can be classified as:

I. **Non- proliferative diabetic retinopathy:**

- Mild
- Moderate
- Severe
- Very severe

II. **Proliferative diabetic retinopathy (PDR)**

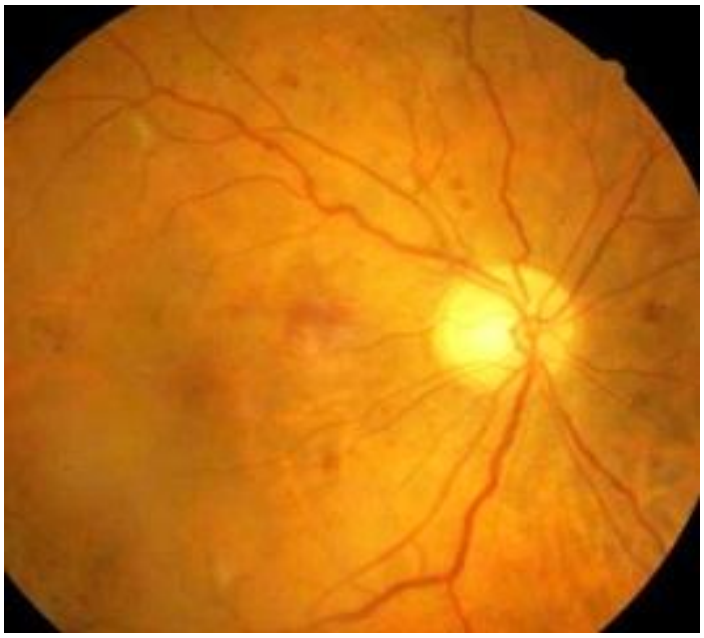
III. **Diabetic maculopathy**

IV. **Advanced diabetic eye disease**

MILD NPDR

MODERATE NPDR

SEVERE NPDR



PDR

The classification used in the **Early Treatment Diabetic Retinopathy Study (Modified Airlie House Classification**) is widely used now-a-days[4].

Non proliferative diabetic retinopathy:

Category	**Clinical features**
No DR	-
Mild (Stage1NPDR)	Atleast one microaneurysm or intraretinal haemorrhage. Hard & soft exudates may or may not be present.
Moderate (Stage2NPDR)	Moderate microaneurysms/ intraretinal haemorrhage. Early mild IRMA. Hard/soft exudates may or may not be present.
Severe (Stage3NPDR)	Any one of the following (4-2-1 rule): Four quadrants of severe microaneurysms/intraretinal haemorrhages. Two quadrants of venous beading. One quadrant of IRMA changes.

Very severe (Stage4NPDR)	Any two of the following: (4-2-1 rule): Four quadrants of severe microaneurysms/intraretinal haemorrhages. Two quadrants of venous beading. One quadrant of IRMA changes.

Proliferative diabetic retinopathy:

Category	**Clinical features**
Mild to moderate	New vessels on the disc (NVD) or new vessels elsewhere (NVE) . But extent insufficient to meet the high risk criteria
High risk	New vessels on the disc (NVD) greater than 1/3 the disc area. Any NVD with vitreous or preretinal haemorrhage. NVE > ½ the disc area with vitreous or preretinal haemorrhage.
Advanced diabetic	Vitreous haemorrhage,

eye disease	tractional RD, tractional retinoschisis, rubeosis iridis.

Diabetic maculopathy:

Clinico – angiographically it can be classified as:

Category	**Clinical features**
Focal	Microaneurysms, hard exudates, haemorrhages, macular edema arranged in circinate pattern.
Diffused	Diffused retinal edema, thickening in posterior pole, few hard exudates.
Ischemic	Marked visual loss + Microaneurysms, hard exudates, few haemorrhages, mild or no macular edema
Mixed	Combined feature of ischemic & exudative maculopathy.

GLAUCOMA IN DIABETES MELLITUS

- **POAG[16,17]:**

Glaucoma is more common in diabetics (5%). But it is only 2% in the general population . In diabetics the risk of glaucoma[22] is higher than in nondiabetic by about 1.6–4.7 times. In the Blue Mountains[32] and Beaver Dam Eye[30] studies it was found that diabetics were twice as likely to have glaucoma as those without.

Microvascular damage from diabetes could impair blood flow to the anterior optic nerve. In the posterior ciliary circulation autoregulation is impaired. This may increase the risk of glaucomatous optic neuropathy. Concomitant cardiovascular risk factors (e.g., hypertension) also increases risk.

But the basic mechanism of increased intraocular pressure appears to be decreased aqueous outflow due to increased resistance at trabecular meshwork[31].

- **NEOVASCULAR GLAUCOMA:**

Proliferative retinopathy is the most common cause of this type of secondary glaucoma[35]. Proliferative diabetic retinopathy causes about 32 and 43% of neovascular glaucoma cases.

PART - II

AIM OF THE STUDY

1. To determine aqueous outflow facility in various stages of diabetic retinopathy.

2. To determine the correlation of aqueous outflow facility in various stages of diabetic retinopathy[24].

3. To determine the correlation of aqueous outflow facility with glycemic control of the patients by analyzing their HbA1C level.

4. To determine the influence of poor glycemic control in diabetic populations with diabetic retinopathy on development of primary open angle glaucoma in them by causing reduction in aqueous outflow facility.

5. To analyse the importance of monitoring tonography for early diagnosis of primary open angle glaucoma in diabetic retinopathy patients ,in addition to measurement of intraocular pressure , monitoring glaucomatous field changes and optic nerve head changes.

INCLUSION CRITERIA:

Clinical criteria for selection of patient for study was:

1. Diabetic patients with and without diabetic retinopathy getting treatment at Department Of Diabetology, Stanley medical college, Chennai.
2. Patients with Bilaterally symmetrical stage of diabetic retinopathy.
3. Patients with no other ocular diseases
4. Patients with no other systemic illness other than diabetes.
5. Patients with open angles in gonioscopy.

EXCLUSION CRITERIA:

1. Patients with other systemic illness other than diabetes mellitus.
2. Patients who underwent previous ocular surgery.
3. Patients with known history of glaucoma with/ without medications.

4. Patients diagnosed to have glaucoma on initial examination at first visit.

5. Patients with secondary glaucoma, narrow angles.

6. Patients with other ocular diseases, dense cataract, opaque media.

7. Patients with high myopia.

8. Patients on systemic medications altering aqueous outflow facility.

9. Patients with no other systemic complications of diabetes mellitus.

10. Patients who had undergone laser treatment for diabetic retinopathy.

MATERIALS AND METHODS

This study was performed on 150 diabetic patients, at Department Of Ophthalmology, Government Stanley Medical college, Chennai, during a period of one year from November 2012 to october 2013.

This study was done in accordance with the rules of ethical committee. All the subjects were explained about the nature of the procedure and an informed consent was obtained.

Materials:

Patients falling inside the inclusion criteria were included for this study.

A total of 150 patients were selected for this study. It included 25 patients in each of the following group- No DR, Stage 1 NPDR, Stage 2 NPDR, Stage3 NPDR, Stage 4 NPDR, PDR.

A detailed medical history was collected from all the participants and they underwent a thorough physical examination, relavant laboratory tests regarding their diabetic status and complete ocular examinations.

<u>The laboratory tests included</u>:

- The estimation of haemoglobin,
- Fasting and post prandial plasma glucose levels,
- Glycated haemoglobin (HbA1C) levels
- Random urine examination for the microalbuminuria[34].

The concentration of HbA1C which was formed through the non-enzymatic attachment of glucose to haemoglobin, was commonly considered to reflect the integrated mean glucose levels over the previous 8–12 weeks, the time period being dictated by the 120 day lifespan of the erythrocyte [38].

<u>The ocular examinations included :</u>

- Assessment of visual acuity,
- Slit-lamp examination of anterior segment,
- Detailed fundus examination,
- Gonioscopy for grading of angles,
- Intraocular pressure (with Goldmann Applanation Tonometer),
- Tonography (with schiotz tonometer),

- Automated perimetry.

APPLANATION TONOMETRY

Automated perimetry

TONOGRAPHY WITH SCHIOTZ TONOMETER

Patients with significantly low aqueous outflow facility were reviewed every 3 months. These patients were subjected to all the needed glaucoma screening investigations and tonography at each visit . Patients glycemic control was also monitored at each visit.

A maximum of 3 review visits were done for these group of patients in this study period. Other patients were asked for review based on their diabetic status.

Patients data at each visit were recorded and compared at each follow up visits.

All the datas were entered in Microsoft Excel and analysed using SSPS PACKAGE(statistical analysis of social science version -16).

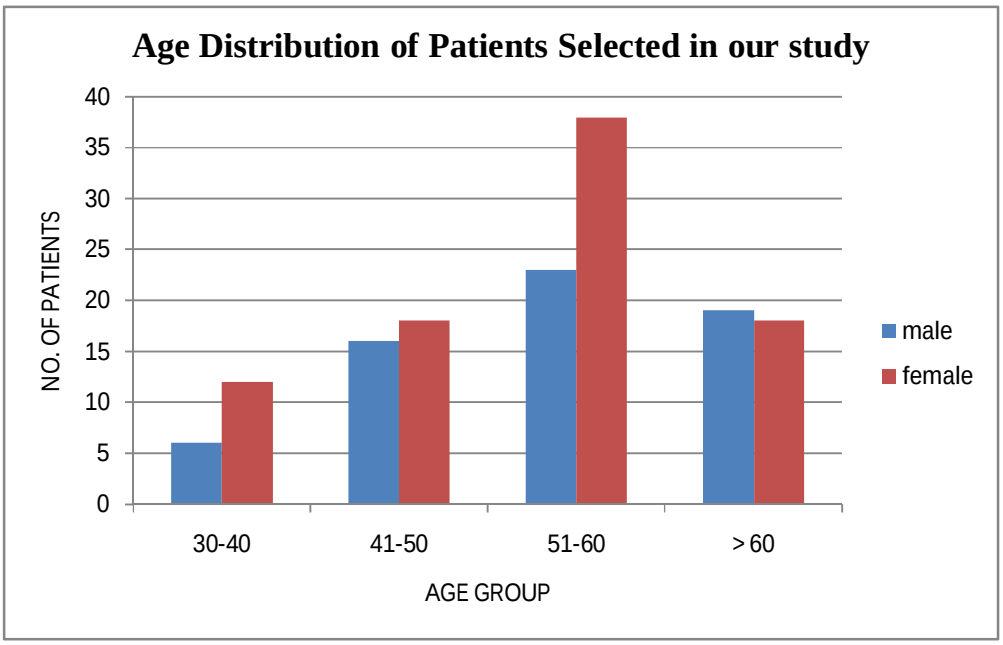
ANOVA test was done to estimate p-value. According to this test P-value was kept significant at < 0.05.

The correlation between HbA1C level and intraocular pressure, and also between HbA1C level and aqueous outflow facility in various stages of diabetic retinopathy was analysed using **Pearson's correlation test** and the datas were plotted in scatter plot diagram. The correlation was assessed based on this 'r ' value.

OBSERVATIONS

Table 1 : Age and sex distribution of patients selected in our study group:

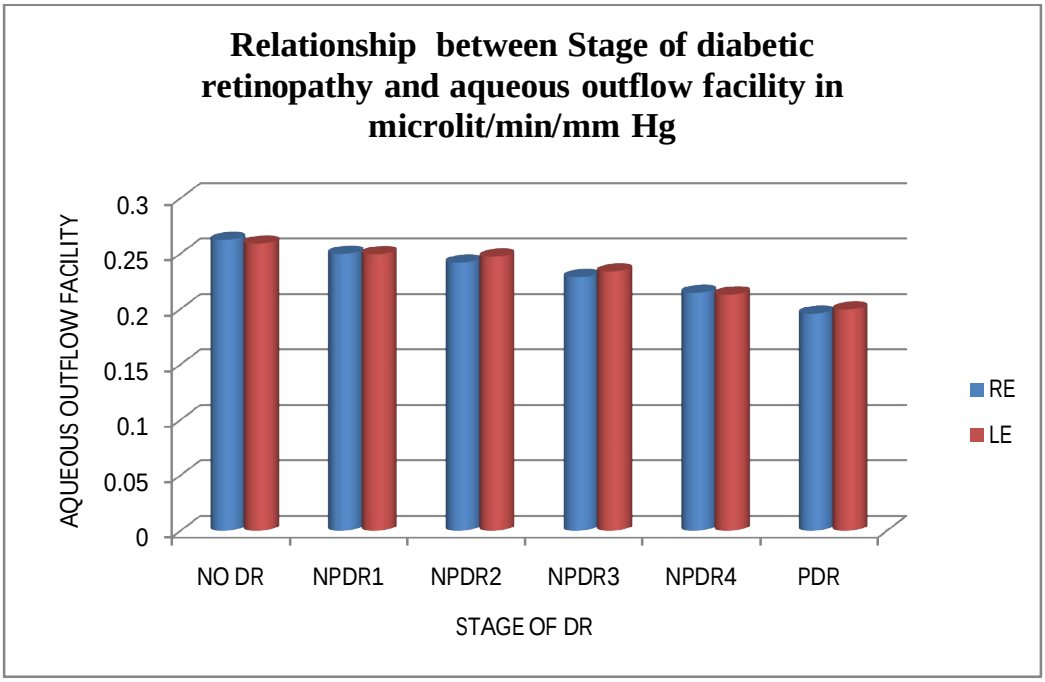
Group	Age in years	Male	female	Total
1	30 – 40	6	12	18
2	41 – 50	16	18	34
3	51 – 60	23	38	61
4	>60	19	18	37
Total		64	86	150
Percentage		42.66%	57.33%	



Maximum number of subjects were in the age group of 51-60 years**.(Table 1)**

TABLE 2: Relationship between Stage of diabetic retinopathy and aqueous outflow facility in microlit/min/mm Hg:

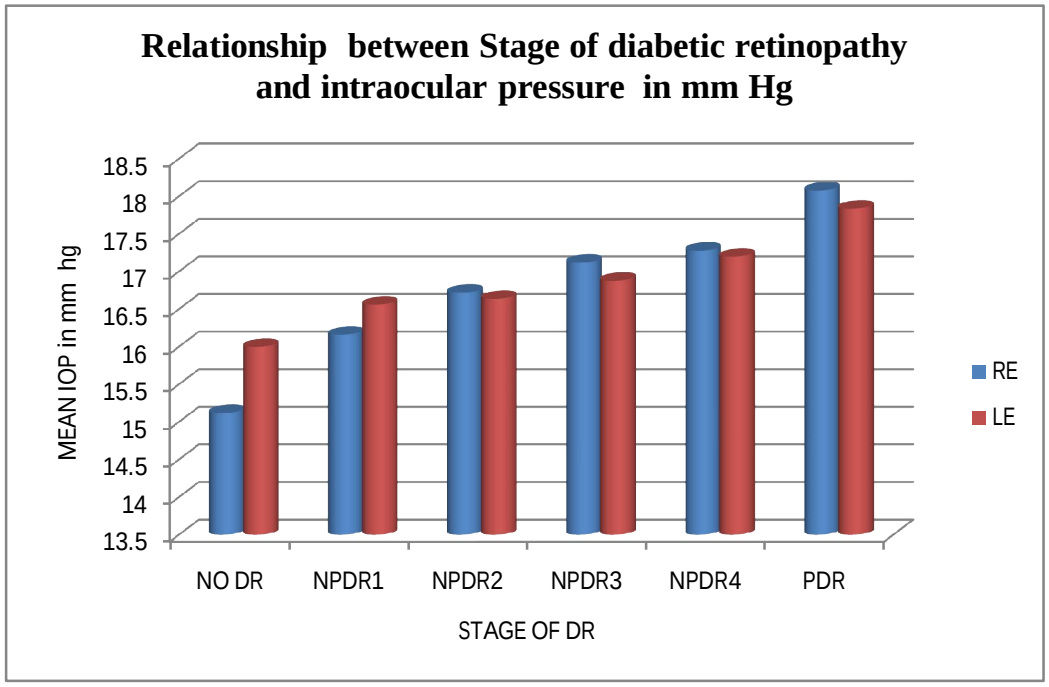
	No DR	Stage1 NPDR	Stage2 NPDR	Stage3 NPDR	Stage4 NPDR	PDR	p-Value*
Average aqueous outflow facility RE	0.2624	0.2500	0.2420	0.2288	0.2148	0.1960	0.000
Average aqueous outflow facility LE	0.2592	0.2496	0.2472	0.2340	0.2132	0.1996	0.000



Average aqueous outflow facility is found to be decreased with advance in the stage of diabetic retinopathy in both the eyes. It is found to be statistically significant (RE p-Value=0.000, LE p-Value= 0.000). **(Table 2)** [*- ANOVA Study was used and p-Value kept significant at < 0.05]

TABLE 3 : Relationship between Stage of diabetic retinopathy and intraocular pressure in mm Hg :

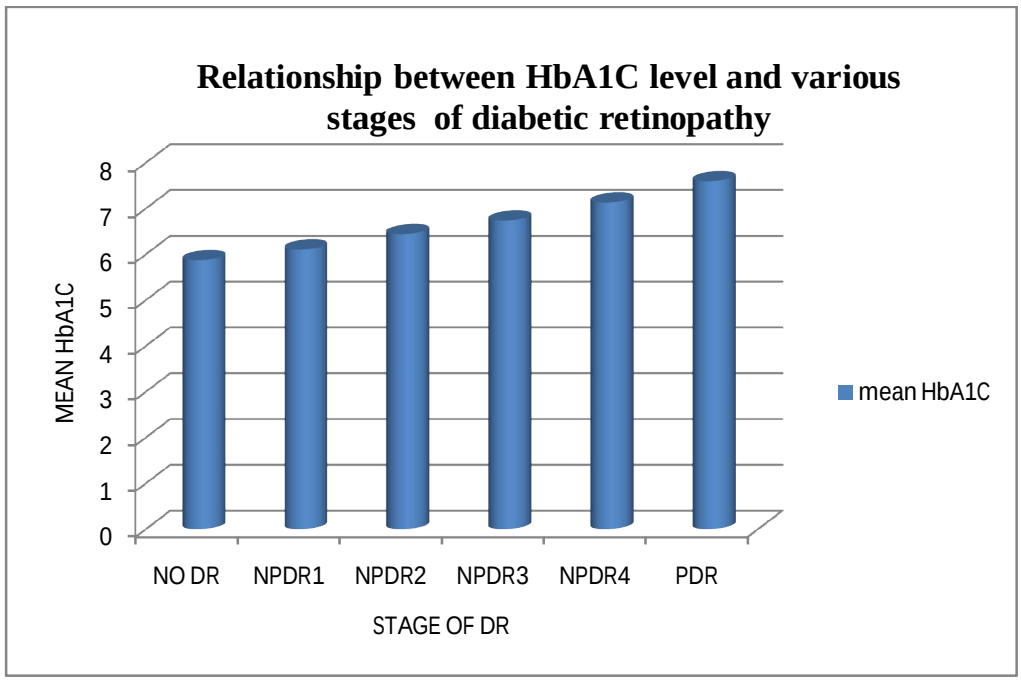
	No DR	**Stage1 NPDR**	**Stage2 NPDR**	**Stage3 NPDR**	**Stage4 NPDR**	**PDR**	**p-Value***
Mean IOP in RE	15.12	16.16	16.72	17.12	17.28	18.08	0.000
Mean IOP in LE	16.00	16.56	16.64	16.88	17.20	17.84	0.006



Mean intraocular pressure was found to be increased with advance in the stage of diabetic retinopathy in both the eyes. It was found to be statistically significant (RE p-Value= 0.000, LE p-Value = 0.006) **(Table 3).** [*- ANOVA Study was used and p-Value kept significant at < 0.05]

TABLE 4: Relationship between HbA1C level and various stages of diabetic retinopathy:

Stage of DR	Mean HbA1C level
No DR	5.866
Stage1 NPDR	6.1
Stage2 NPDR	6.44
Stage3 NPDR	6.732
Stage4 NPDR	7.128
PDR	7.592
p-Value*	0.000

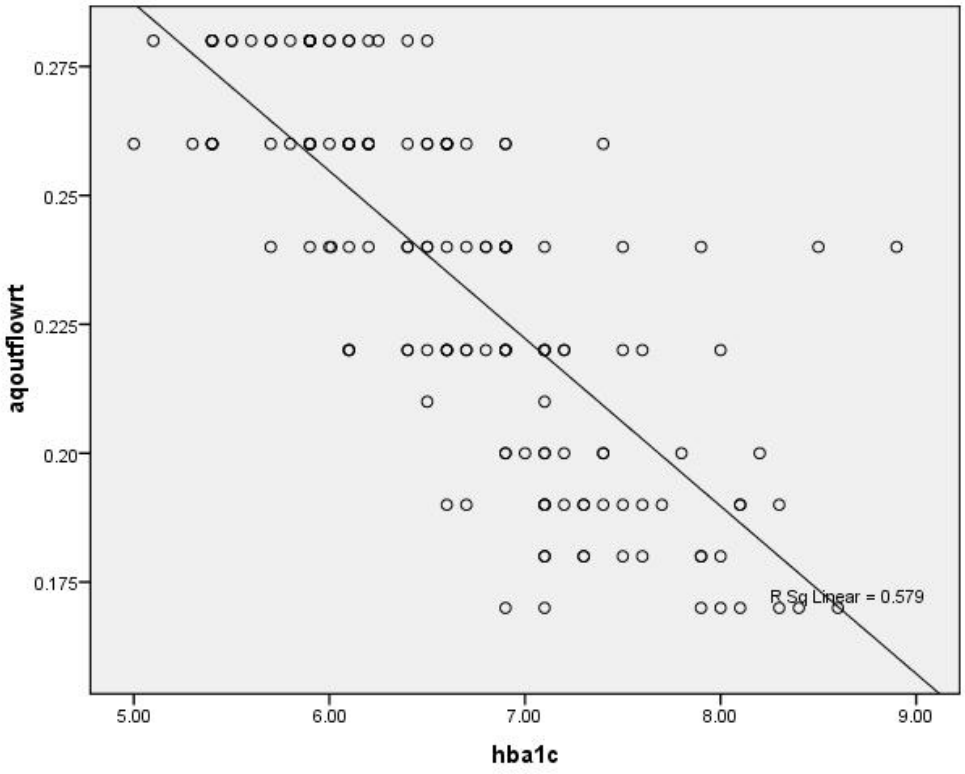


Mean HbA1C level was found to be high with advance in the stage of diabetic retinopathy. It was found to be statistically significant (p-Value= 0.000) **(Table 4)**. [*- ANOVA Study was used and p-Value kept significant at < 0.05]

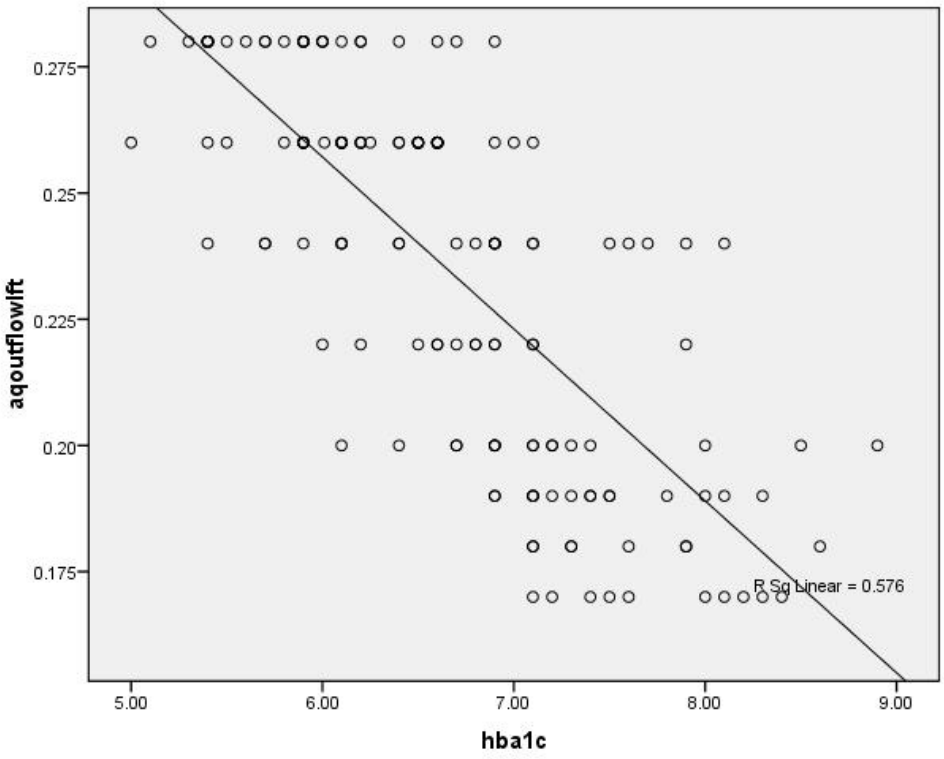
TABLE 5: Association between Mean HbA1C and aqueous outflow facility in microlit/min/ mm Hg in various stages of DR:

Stage of DR	No DR	Stage1 NPDR	Stage2 NPDR	Stage3 NPDR	Stage4 NPDR	PDR	r- Value p-Value*
Mean HbA1C level	5.866	6.1	6.44	7.32	7.128	7.592	
Average aqueous outflow facility RE	0.2624	0.2500	0.2420	0.2288	0.2148	0.1960	r= -0.761 p=0.000
Average aqueous outflow facility LE	0.2592	0.2496	0.2472	0.2340	0.2132	0.1996	r= -0.759 p= 0.000

Scatter plot showing correlation between Mean HbA1C and aqueous outflow facility in various stages of DR in Right Eye :



Scatter plot showing correlation between Mean HbA1C and aqueous outflow facility in various stages of DR in Left Eye :

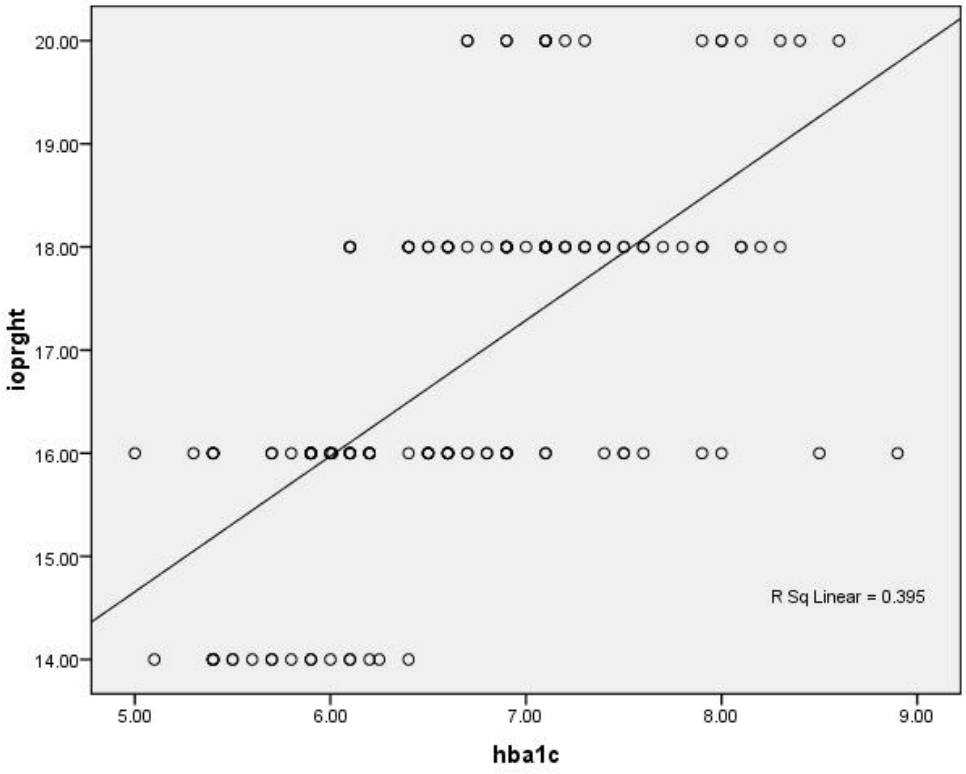


Average aqueous outflow facility was found to be decreased with increase in mean HbA1C level in various stages of diabetic retinopathy in both eyes. (RE r= -0.761, p-Value= 0.000, LE r= -0.759, p-Value= 0.000) **(Table 5)** [*- ANOVA Study was used and p-Value kept significant at < 0.05]

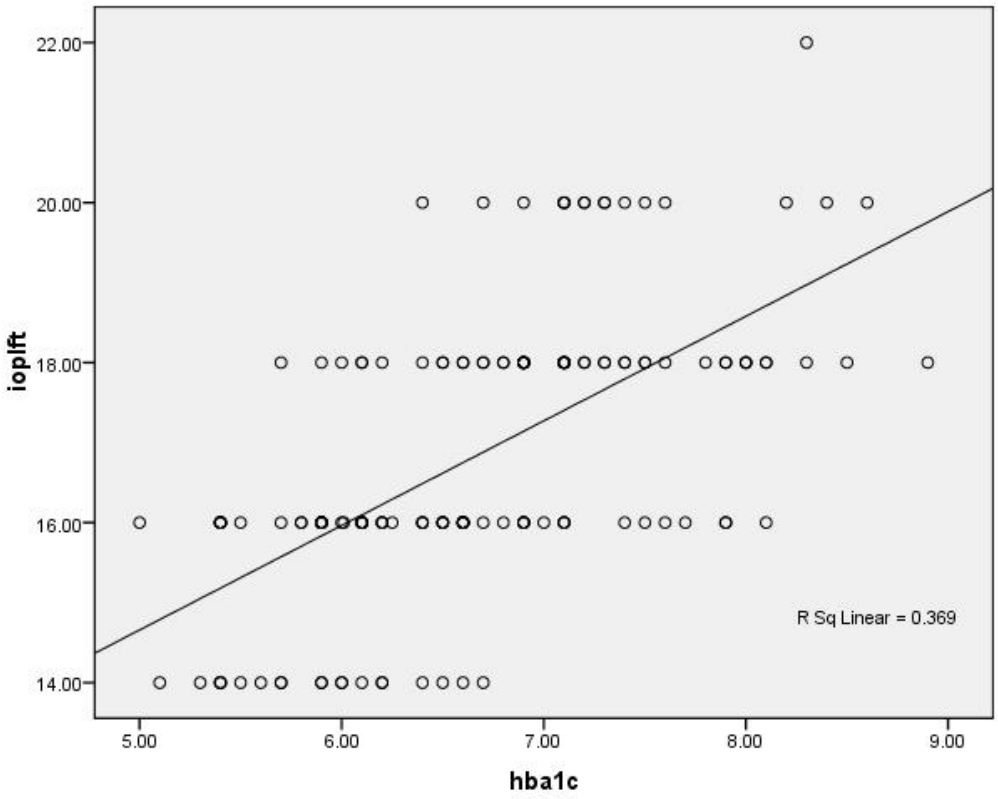
TABLE 6: Association between mean HbA1C level and IOP in mm Hg in various stages of DR:

Stage of DR	**No DR**	**Stage1 NPDR**	**Stage2 NPDR**	**Stage3 NPDR**	**Stage4 NPDR**	**PDR**	**r- Value p-Value***
Mean HbA1C level	5.866	6.1	6.44	7.32	7.128	7.592	
Mean IOP in RE	15.12	16.16	16.72	17.12	17.28	18.08	r= 0.628 p= 0.000
Mean IOP in LE	16.00	16.56	16.64	16.88	17.20	17.84	r= 0.608 p= 0.000

Scatter plot showing correlation between Mean HbA1C and Mean IOP in various stages of DR in Right Eye :



Scatter plot showing correlation between Mean HbA1C and Mean IOP in various stages of DR in Left Eye :



Average IOP was found to be increased with increase in mean HbA1C level in various stages of diabetic retinopathy in both the eyes. It was found to be statistically significant. (RE r= 0.628, p-Value= 0.000, LE r= 0.608, p-Value= 0.000).**(Table 6)** [*- ANOVA Study was used and p-Value kept significant at < 0.05]

TABLE 7:Association of Mean ocular rigidity in relation to retinal status:

	No DR	**Stage1 NPDR**	**Stage2 NPDR**	**Stage3 NPDR**	**Stage4 NPDR**	**PDR**	**p-Value***
Mean ocular rigidity RE	0.0217	0.0216	0.0216	0.0217	0.0216	0.0216	0.783
Mean ocular rigidity LE	0.0217	0.0216	0.0216	0.0216	0.0216	0.0216	0.814

There was no significant difference in ocular rigidity in various stages of diabetic retinopathy in both eyes. (RE p-Value= 0.783, LE p-Value= 0.814).**(Table 7)** [*- ANOVA Study was used and p-Value kept significant at < 0.05]

RESULTS

A total of 150 subjects were recruited in our study. They were in the age group ranging from 30 yrs and above.

All the recruited subjects were within the inclusion criteria for our study.

The following were the results of our study:

Average aqueous outflow facility was found to be decreased with advance in the stage of diabetic retinopathy in both the eyes. It was found to be statistically significant (RE p-Value=0.000, LE p-Value= 0.000).[TABLE 2]

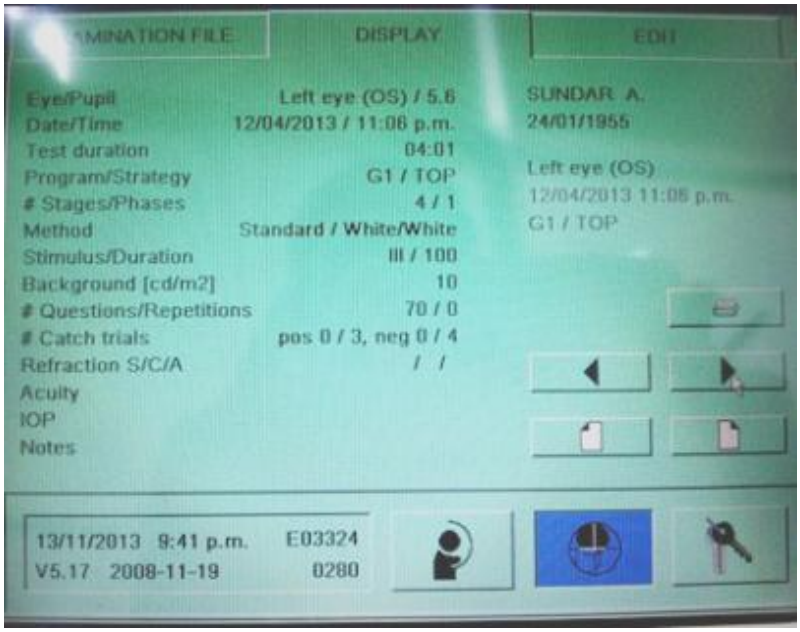
Mean intraocular pressure was found to be increased with advance in the stage of diabetic retinopathy in both the eyes. It was found to be statistically significant (RE p-Value= 0.000, LE p-Value = 0.006). [TABLE 3]

Mean HbA1C level was found to be high with advance in the stage of diabetic retinopathy. It was found to be statistically significant (p-Value= 0.000). Average aqueous outflow facility was found to be decreased with increase in mean HbA1C level in various stages of

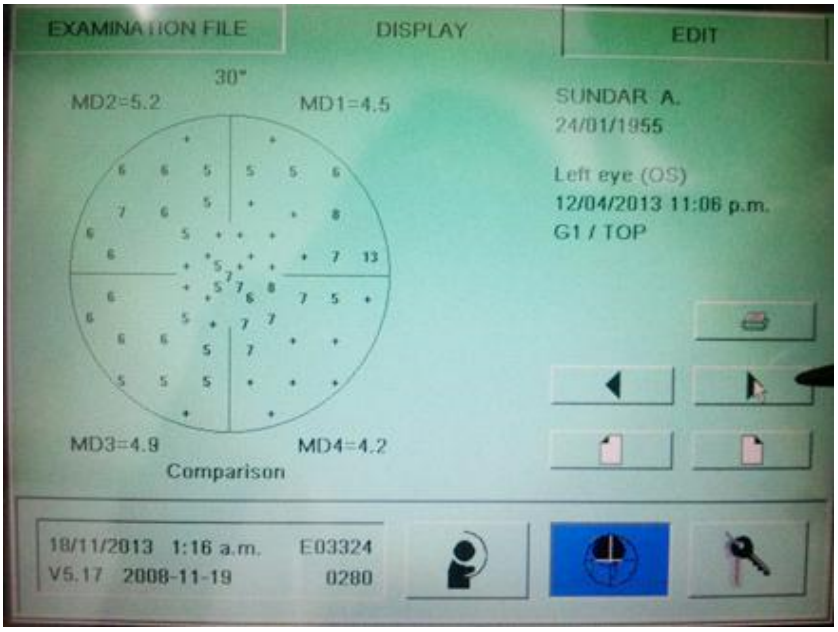
diabetic retinopathy in both eyes. (RE r= -0.761, p-Value= 0.000, LE r= -0.759, p-Value= 0.000) . [TABLE 4]

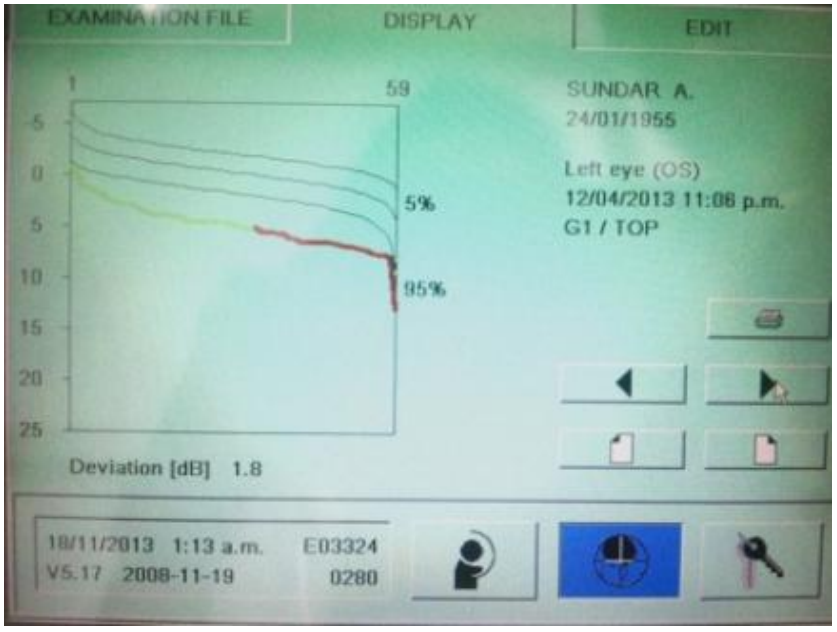
OPTIC NERVE HEAD CHANGES IN GLAUCOMA

PERIMETRY WITH GLAUCOMATOUS FIELD DEFECTS



PERIMETRY WITH GLAUCOMATOUS FIELD DEFECTS





Average IOP was found to be increased with increase in mean HbA1C level in various stages of diabetic retinopathy in both the eyes. It was found to be statistically significant. (RE r=0.628, p-Value= 0.000, LE r= 0.608, p-Value= 0.000). [TABLE 5]

Patients were asked for follow up based on their findings.

Among the 150 patients 43 patients had significantly reduced aqueous outflow facility at their first visit examination. Hence they were followed up every 3 months.

Among 43 patients advised follow up , 22 patients attended regular follow up of maximum 3 visits every 3 months in this one year study period.

They underwent the needed ocular and systemic examinations in all the 3 visits and the results were compared . 21 patients did not attend complete follow up as per advice.

At the end of this one year period 2 of the 22 patients were found to develop early glaucomatous field changes in their visual field. They were also found to have continued poor glycemic control (HbA1C > 6.5), significantly low aqueous outflow facility and intraocular pressure

slightly increased compared to the initial recording. [FOLLOW UP CHART IN ANNEXURE]

DISCUSSION

Our study reveals that aqueous outflow facility is getting reduced significantly with advance in the stage of diabetic retinopathy and also with poor glycemic control .

Our study also reveals that intraocular pressure is at a comparatively higher level in patients with poor glycemic controls (HbA1C > 6.5) and also with advance in the stage of diabetic retinopathy.

Beaver Dam Study[30] in 1994 concluded that presence of open angle glaucoma is increased in people with late onset diabetes.

Los angeles Latino Eye Study in 2008 concluded that presence of Type2DM was associated with increased risk of having open angle glaucoma.

Arora Vn,Prasad Vn in 1989 have also reported that IOP was comparatively increased in patients with diabetes mellitus with diabetic retinopathy than in patients without diabetic retinopathy. Our study also concludes that intraocular pressure is higher in diabetic patients with advance in stage of diabetic retinopathy.

Matsuoka et al[43], in 2012 reported that there is significantly higher IOP in diabetic patients with advance in the stage of diabetic retinopathy and also with poor glycemic control. Our study also concludes the same.

	Mean Iop in the following stage of DR	**Matsuoka et al study**	**Arora Vn study**	**Our study**	
				RE	**LE**
Observations	No DR	15.6	18.17	15.12	16.00
	Mild to Moderate NPDR	15.7	20.98 to 21.99	16.16 to 16.72	16.56 to 16.64
	Severe NPDR	15.00	22.18	17.12	16.88
	PDR	18.00	15.98	18.00	17.84
Inference		Mean IOP is significantly increased in patients with diabetic retinopathy (p-value < 0.001	Mean IOP is increased in patients with advance in stage of NPDR	Mean IOP was significantly higher with advance in the stage of diabetic retinopathy in both eyes. (RE p-value = 0.000, LE p -Value = 0.006)	

Anandalakshmi et al[8] in 2011 reported that mean IOP was higher in patients with DM and also in patients with poor glycemic control. Our study also concludes that IOP is significantly higher in patients with poor glycemic control.

	Anandalakshmi et al study	**Our study**	
		RE	**LE**
Observation	Mean Iop with mean HbA1C Of 6.00 } 17.32	Mean Iop with mean HbA1C Of 5.866 }=15.12	Mean Iop with mean HbA1C Of 5.866 }= 16.00
	Iop with mean HbA1C Of 7.42 } 20.98	Iop with mean HbA1C Of 7.592 }= 18.08	Iop with mean HbA1C Of 7.592 }= 17.84
	p- Value = 0.001 r- Value = 0.82	p- Value = 0.000 r- value = 0.628	p- value = 0.000 r – value = 0.608
Inference	Mean IOP is diabetic patients was found to be increased with increase in mean HbA1C level (ie. with poor glycemic control).		

The reason for association between POAG and diabetes mellitus is still to be completely elucidated. It has been reported in as early as 1984 by Davies et al that glucose level in aqueous humor is higher in patients with diabetes mellitus.

It has been found that the high glucose level in aqueous humor in patients with diabetes mellitus leads to fibronectin upregulation in trabecular meshwork[20] .

This compromises trabecular meshwork cellularity[50], resiliency and alters its structural content. Hence aqueous outflow facility is reduced at Trabecular Meshwork level leading to raised IOP and finally patients will be at risk of POAG[33].

Arora Vn,Prasad Vn in 1989 have reported that aqueous outflow facility is reduced in patients with advance in stage of diabetic retinopathy, especially (NPDR > PDR). Our study also concludes that aqueous outflow is significantly reduced with advance in the stage of diabetic retinopathy.

	Mean Aqueous outflow facility in microlit/min/ mm Hg in the following stages of DR	**Arora Vn study**	**Our study**	
			RE	**LE**
Observations	No DR	0.265	0.2624	0.2592
	Mild DR	0.201	0.2500	0.2496
	Moderate DR	0.231	0.2420	0.2472
	Severe DR	0.220	0.2288	0.2340
	PDR	0.315	0.1960	0.1996
Inference		Facility of aqueous outflow is getting reduced with advance in stage of NPDR	Facility of aqueous outflow is getting reduced significantly with advance in the stage of DR (RE p-Value = 0.000, LE p-Value = 0.000)	

Also due to disturbance in microcirculation in Diabetes Mellitus[39], these patients are at early risk of ONH damage[40,41]. Also Retinal Ganglion Cell death occurs earlier in them due to altered metabolism.

Our study found that when patients with significantly reduced aqueous outflow facility with poor glycemic control were regularly followed up over this one year period, 2 patients of the 22 patients developed early glaucomatous field defects at follow up visits compared to normal fields in their initial visits. They were also observed to have continued poor glycemic control and reduced aqueous outflow facility in this study period.

Hence our study reveals that monitoring aqueous outflow facility also, while screening for early detection of primary open angle glaucoma will be a useful guide along with other parameters of glaucoma evaluation which is routinely done.

CONCLUSION

Glaucoma is one of the preventable causes of irreversible visual impairment, if diagnosed and treated at earlier stages[37]. Diabetes has been shown to be one of the risk factors for POAG.

Hence early screening of diabetic patients with appropriate techniques, will help to detect people at risk to develop POAG .Hence they can be followed up regularly with appropriate treatment when needed.

Since aqueous outflow facility is also one of the important component in determining the intraocular pressure , it will be more appropriate to measure it , especially in diabetics.

Tonography with schiotz tonometer is a **simple, inexpensive** and **non- invasive** procedure. It can be done as an **outpatient procedure**.

It can be done even in peripheral health centres to detect people at risk to develop POAG so that they can be referred in time to higher centres for early treatment and prevention of glaucomatous optic nerve damage.

This study has revealed that performing tonography and detecting patients with significantly low aqueous outflow facility can be a guidance for early diagnosis of development of POAG rather than relying on IOP alone .

Hence tonography can be used as one of the examination techniques along with IOP measurement , optic nerve head evaluation and perimetry, for early identification of risk of developing POAG due to reduced aqueous outflow facility in diabetic patients with advanced stages of diabetic retinopathy.

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PROFORMA

Name: Age: Sex:

OP/IP no:

Presenting illness:

Past history:

Diabetes history-

Treatment history-

H/O other medical illness-

Family history:

Diabetes- Glaucoma-

Details of examination:

General examination:

Ocular examination:

	Right eye	left eye
1.Eyelids		
2.Eyelashes		
3.Eom		
4.Conjunctiva		
5.Cornea		

6.Pupil

7.Iris

8. Anterior chamber

9.Lens

10.Visual acuity:

Distance

Near

11. Colour vision

12. Retinoscopy:

13. PMT:

14. Fields with automated perimetry:

15. IOP with applanation tonometer :

16. Gonioscopy:

17.Fundus findings:

Direct ophthalmoscope:

Indirect ophthalmoscope:

+90 D examination:

Aqueous outflow measurement with schiotz tonometer:

	RE 5.5 gms	**LE 5.5 gms**	**RE 7.5 gms**	**LE 7.5 gms**
Scale reading at 0 mts				
Scale reading at 4 mts				
Facility of aqueous outflow				

Ocular rigidity based on Modified Fridenwald's Nomogram:

	RE	**LE**
IOP with 5.5 gms		
IOP with 10.00 gms		
Ocular rigidity		

Inference: **RE** **LE**

1. Stage of diabetic retinopathy:
2. Mean Intraocular pressure:
3. Mean Aqueous outflow facility:
4. Ocular rigidity:

Advice :

ABBREVIATIONS

S.NO	-	SERIAL NUMBER
DM	-	DIABETES MELLITUS
DR	-	DIABETIC RETINOPATHY
AQ	-	AQUEOUS
IOP	-	INTRAOCULAR PRESSURE
RE	-	RIGHT EYE
LE	-	LEFT EYE
M	-	MALE
F	-	FEMALE
YRS	-	YEARS
MTH	-	MONTHS
OHA	-	ORAL HYPOGLYCEMIC AGENTS
ISN	-	INSULIN
NPDR	-	NON PROLIFERATIVE DIABETIC RETINOPATHY
NPDR1	-	MILD NON PROLIFERATIVE DIABETIC RETINOPATHY
NPDR2	–	MODERATE NON PROLIFERATIVE DIABETIC RETINOPATHY
NPDR3	–	SEVERE NON PROLIFERATIVE DIABETIC RETINOPATHY

NPDR4	-	VERY SEVERE NON PROLIFERATIVE DIABETIC RETINOPATHY
PDR	-	PROLIFERATIVE DIABETIC RETINOPATHY
CSME	-	CLINICALLY SIGNIFICANT MACULAR EDEMA
FCSME	-	FOCAL CLINICALLY SIGNIFICANT MACULAR EDEMA
DCSME	-	DIFFUSED CLINICALLY SIGNIFICANT MACULAR EDEMA
IOP	-	INTRAOCULAR PRESSURE
V	-	VISIT
LFU	–	LOST FOLLOW UP
M LIT	–	MICROLITRE
MM HG	–	MILLIMETRE OF MERCURY
YRS	–	YEARS
AQ	–	AQUEOUS
POAG	-	PRIMARY OPEN ANGLE GLAUCOMA

MASTER CHART

S .NO	NAME	Age in yrs/ Sex	Duration of DM from Diagnosis	Treatment	Stage of DR		AQ outflow Facility in mlit/min/mm hg		IOP in mm hg		Ocular Rigidity		HbA1c
					RE	LE	RE	LE	RE	LE	RE	LE	
1	dhanalakshmi	46/F	32 YRS	OHA	NPDR4	NPDR4	0.19	0.18	18	20	0.022	0.0218	7.3
2	kasthuri	65/F	1 MTH	OHA	NPDR1	NPDR1	0.22	0.2	18	18	0.0214	0.0214	6.9
3	gunasekar	50/M	16 YRS	INSULIN	PDR+CSME	PDR	0.17	0.19	20	18	0.0216	0.022	8.1
4	babu	63/M	2 WKS	INSULIN	NPDR4+CSME	NPDR4	0.26	0.24	16	16	0.022	0.022	6.9
5	rajaram	45/M	17 YRS	OHA+ISN	PDR	PDR	0.19	0.19	18	18	0.022	0.0218	7.4
6	kandasamy	46/M	15 YRS	OHA+ISN	PDR	PDR	0.22	0.17	16	18	0.0216	0.0216	8
7	senthil	40/M	10 YRS	OHA+ISN	PDR+CSME	PDR+CSME	0.18	0.22	18	16	0.0217	0.0217	7.9
8	jaithan bee	45/F	13 YRS	OHA	NPDR2+CSME	NPDR2+CSME	0.22	0.22	18	18	0.0215	0.0215	6.8
9	sunitha bee	45/F	10 YRS	OHA	NPDR3+CSME	NPDR3+CSME	0.24	0.22	16	18	0.022	0.022	6.8
10	kokila	54/F	15 YRS	OHA	NPDR3+CSME	NPDR3+CSME	0.22	0.24	18	16	0.0216	0.0216	7.1
11	kurshith banu	53/F	10 YRS	OHA+ISN	NPDR4+FCSME	NPDR4+DCSME	0.26	0.24	16	16	0.0217	0.0215	6.7
12	kasthuri	60/F	7 YRS	OHA	NPDR2+CSME	NPDR2+CSME	0.24	0.24	16	16	0.0215	0.0215	6.9
13	anthony	40/F	3 MTHS	OHA	NPDR1	NPDR1	0.22	0.26	18	16	0.0215	0.0215	6.4
14	meenakshi	37/F	10 YRS	OHA+ISN	PDR	PDR	0.19	0.17	18	18	0.022	0.022	8.1
15	sathyavathy	60/F	20 YRS	OHA	NPDR2+CSME	NPDR2+CSME	0.22	0.24	18	16	0.0216	0.0216	6.4
16	kalikambal	70/F	3 YRS	OHA	NPDR2	NPDR2	0.24	0.26	16	16	0.0217	0.0216	6.2
17	sujatha	39/F	1 WK	0HA	NPDR1	NPDR1	0.22	0.2	18	20	0.0214	0.0214	6.7
18	madeshwaran	30/M	16 YRS	OHA	NPDR2+CSME	NPDR2+CSME	0.22	0.26	18	18	0.0215	0.0215	6.9
19	devikha	52/F	12 YRS	OHA+ISN	PDR	PDR	0.19	0.19	18	18	0.0214	0.0214	8.3
20	kamala	50/F	4 YRS	OHA	NPDR2	NPDR2	0.26	0.24	16	16	0.022	0.022	6.1
21	meena	50/F	13 YRS	OHA+ISN	NPDR4	NPDR4	0.18	0.18	18	18	0.0217	0.0217	7.3
22	kumar	39/M	7 YRS	OHA	NPDR2	NPDR2	0.26	0.22	16	18	0.0214	0.0214	6.2
23	rajendran	49/M	12 YRS	OHA	NPDR3+CSME	NPDR3+CSME	0.22	0.24	18	16	0.0216	0.0216	7.1
24	sangeetha	35/F	7 YRS	OHA+ISN	PDR+VH	PDR+VH	0.22	0.24	16	16	0.0216	0.0216	7.5
25	roopavathy	60/F	20 YRS	OHA	NPDR3+CSME	NPDR3+CSME	0.24	0.26	16	14	0.0215	0.0217	5.9
26	gunasekar	50/M	17 YRS	INSULIN	PDR+CSME	PDR+CSME	0.17	0.18	20	18	0.0216	0.0216	7.9
27	dhanalakshmi	60/F	13 YRS	INSULIN	NPDR3	NPDR3	0.2	0.19	18	20	0.0217	0.0217	7.4
28	poochendu	65/F	16 YRS	INSULIN	PDR+CSME	PDR+CSME	0.18	0.18	18	18	0.0215	0.0215	7.9
29	deepak kadir	60/M	10 YRS	OHA+ISN	NPDR3	NPDR3	0.2	0.2	18	18	0.0214	0.0214	6.9
30	vahith	62/M	5 YRS	OHA	NPDR3	NPDR3	0.26	0.26	16	16	0.0216	0.0217	6.6
31	uma	55/F	5 YRS	OHA	NPDR3	NPDR3	0.26	0.2	16	18	0.0217	0.0216	7.4

S .NO	NAME	Age in yrs/ Sex	Duration of DM from Diagnosis	Treatment	Stage of DR		AQ outflow Facility in mlit/min/mm hg		IOP in mm hg		Ocular Rigidity		HbA1c
					RE	LE	RE	LE	RE	LE	RE	LE	
32	maariyama	60/F	8 YRS	OHA	NPDR3	NPDR3	0.2	0.22	18	18	0.022	0.022	7.1
33	kowsalya	52/F	15 YRS	OHA	NPDR3	NPDR3	0.19	0.19	20	20	0.0216	0.0216	7.2
34	Vasantha kumar	54/M	1.5 YRS	OHA	NPDR2	NPDR2	0.22	0.26	18	16	0.0214	0.0214	6.6
35	beenali raj	49/M	7 YRS	OHA	NPDR3	NPDR3	0.2	0.19	18	18	0.0217	0.0217	6.9
36	noorjahan	39/F	12 YRS	OHA+ISN	NPDR3	NPDR3	0.19	0.19	18	18	0.0215	0.0217	7.1
37	mary	62/F	3 YRS	OHA	NPDR2	NPDR2	0.22	0.22	18	18	0.0214	0.0214	6.9
38	maragatham	57/F	11 YRS	OHA	NPDR3	NPDR3	0.26	0.26	16	16	0.0217	0.0216	5.9
39	yasodha	47/F	5 YRS	OHA	NPDR1	NPDR1	0.28	0.28	16	16	0.0214	0.0214	5.9
40	reeta	60/F	4 YRS	OHA	NPDR1	NPDR1	0.28	0.26	16	18	0.0216	0.0216	6.5
41	rani	54/F	7 YRS	OHA+ISN	PDR	PDR	0.17	0.17	20	20	0.0215	0.0216	8.4
42	munusamy	40/M	5 YRS	OHA	NO DR	NO DR	0.28	0.24	16	16	0.022	0.0221	5.4
43	vaiayanthi	35/F	11.5 YRS	OHA	NPDR3	NPDR3	0.26	0.28	16	18	0.0214	0.0214	6.9
44	sundar	55/M	12 YRS	OHA+ISN	NPDR4	NPDR4	0.19	0.18	16	18	0.0215	0.0216	7.1
45	Vanishree	48/F	7 YRS	OHA+ISN	PDR	PDR	0.19	0.17	18	20	0.0216	0.0216	7.6
46	pichai	56/M	2 YRS	OHA	NPDR1	NPDR1	0.22	0.26	16	16	0.022	0.0219	6.1
47	selvam	64/M	1 YR	OHA	NPDR3	NPDR3	0.21	0.26	18	16	0.0216	0.0216	6.5
48	ravindran	49/M	6YRS	OHA	NO DR	NO DR	0.28	0.24	16	18	0.0217	0.0217	5.9
49	arputham	67/M	1 YR	OHA	NPDR1	NPDR1	0.26	0.26	16	16	0.0215	0.0216	5.8
50	ramaiyah	58/M	8 YRS	OHA	NPDR2	NPDR2	0.22	0.2	18	20	0.0216	0.0216	7.2
51	shenbagam	54/F	14 YRS	OHA	NPDR2	NPDR2	0.22	0.22	18	20	0.0214	0.0214	7.1
52	muthumani	51/M	11 YRS	OHA	NPDR1	NPDR1	0.28	0.28	14	14	0.0217	0.0217	5.7
53	parvathy	48/F	5 YRS	OHA	NPDR1	NPDR1	0.24	0.24	18	18	0.0216	0.0216	6.4
54	veeraiyah	60/M	13.5 YRS	OHA+ISN	NPDR4	NPDR4	0.18	0.2	18	18	0.0217	0.0216	7.3
55	anushya	53/F	10YRS	OHA+ISN	NPDR4	NPDR4	0.24	0.19	16	18	0.0214	0.0214	6.9
56	vimaladevi	59/F	5 YRS	OHA	NPDR2	NPDR2	0.24	0.22	18	18	0.0217	0.0217	6.9
57	maari	58/M	5 YRS	OHA	NPDR1	NPDR1	0.26	0.2	16	18	0.0215	0.0215	6.1
58	maariyaman	60/M	8 YRS	OHA	NPDR1	NPDR1	0.28	0.28	14	14	0.0216	0.0216	5.9
59	kannan	60/M	5 YRS	OHA	NPDR2	NPDR2	0.26	0.28	16	14	0.0215	0.0215	6.2
60	shanmugam	49/M	3 YRS	OHA	NPDR1	NPDR1	0.19	0.2	20	20	0.0216	0.0216	7.1
61	narayanan	50/M	5 MTH	OHA	NPDR1	NPDR1	0.22	0.22	18	18	0.0217	0.0217	6.5
62	padma	50/F	1 YR	OHA	NPDR1	NPDR1	0.22	0.26	18	16	0.0216	0.0215	6.6
63	raman	65/M	2 YRS	OHA	NPDR2+CSME	NPDR2+CSME	0.26	0.26	16	16	0.022	0.022	6.6
64	sussela	54/F	10 YRS	OHA	NPDR2+CSME	NPDR2+CSME	0.19	0.2	20	18	0.0217	0.0217	6.7
65	devaki	55/F	3 YRS	OHA	NPDR1	NPDR1	0.22	0.22	20	18	0.0215	0.0215	6.7

S .NO	NAME	Age in yrs/ Sex	Duration of DM from Diagnosis	Treatment	Stage of DR		AQ outflow Facility in mlit/min/mm hg		IOP in mm hg		Ocular Rigidity		HbA1c
					RE	LE	RE	LE	RE	LE	RE	LE	
66	lakshmi	58/F	6 YRS	OHA	NPDR1	NPDR1	0.28	0.26	14	16	0.0215	0.0216	6.1
67	shanthi	57/F	5 YRS	OHA	NPDR3	NPDR3	0.19	0.19	20	20	0.0214	0.0214	7.3
68	gowri	57/F	7 YRS	OHA	NPDR1	NPDR1	0.28	0.28	14	16	0.0216	0.0216	5.8
69	kamathilakam	62/F	5 YRS	OHA	NPDR1	NPDR1	0.26	0.28	14	14	0.022	0.022	5.4
70	lakshmi	46/F	10 YRS	OHA	NPDR3	NPDR3	0.19	0.26	18	16	0.0217	0.0216	6.6
71	michael raj	67/M	13 YRS	OHA	NPDR1	NPDR1	0.28	0.26	14	16	0.0215	0.0215	5.5
72	vellathai	70/F	1.5 YRS	OHA	NPDR4	NPDR4	0.18	0.17	20	18	0.0216	0.0216	7.1
73	vishalatchi	53/F	8 YRS	OHA	NPDR4	NPDR4	0.2	0.18	18	18	0.0216	0.0216	7.1
74	ramanappan	76/M	1 YRS	OHA	NPDR2	NPDR2	0.24	0.26	16	16	0.0217	0.0217	6.6
75	desammal	60/F	2 YRS	OHA	NO DR	NO DR	0.26	0.28	16	14	0.0216	0.0216	6
76	subhulakshmi	53/F	2.5 YRS	OHA	NPDR2+CSME	NPDR2+CSME	0.28	0.28	14	16	0.0217	0.0217	5.9
77	padmavathy	60/F	4 YRS	OHA	NPDR2+CSME	NPDR2+CSME	0.28	0.28	14	14	0.0216	0.0216	5.4
78	vairamani	50/M	1 YRS	OHA	NPDR1	NPDR1	0.26	0.26	16	16	0.0214	0.0214	6.1
79	uma	48/F	3 YRS	OHA	NPDR1	NPDR1	0.24	0.22	16	18	0.022	0.022	6
80	kalyani	52/F	7 YRS	OHA	NPDR2	NPDR2	0.28	0.28	14	14	0.0217	0.0217	5.6
81	senjiappan	40/M	9MTH	OHA	NO DR	NO DR	0.26	0.28	14	14	0.0216	0.0216	5.7
82	rajeshwari	60/F	5 YRS	OHA	NPDR2	NPDR2	0.26	0.28	16	16	0.0217	0.0216	5.9
83	dhanalakshmi	52/F	3.1 YRS	OHA+ISN	NPDR2	NPDR2	0.22	0.26	18	16	0.0215	0.0215	6.1
84	mallika	55/F	8 YRS	OHA	NPDR4	NPDR4	0.24	0.24	16	16	0.022	0.022	6.9
85	amudha	51/F	6 YRS	OHA	NPDR2	NPDR2	0.26	0.26	16	16	0.0216	0.0216	6.2
86	parvathy	68/F	6 MTH	OHA	NPDR4	NPDR4	0.18	0.17	18	20	0.0215	0.0215	7.5
87	kalaiarasar	50/M	10 YRS	OHA	NPDR2	NPDR2	0.22	0.26	18	16	0.0217	0.0216	6.6
88	krishnaraj	65/M	10.3 YRS	OHA	NPDR4	NPDR4	0.26	0.28	16	14	0.0214	0.0215	6.6
89	selvi	65/F	4 YRS	OHA	NPDR3	NPDR3	0.26	0.26	16	16	0.0216	0.0216	5.9
90	indirani	70/F	10 YRS	OHA	NPDR4	NPDR4	0.2	0.26	18	16	0.022	0.022	7
91	dhamayanthi	35/F	4 YRS	OHA+ISN	PDR	PDR	0.24	0.2	16	18	0.0216	0.0217	8.5
92	padmavathy	40/F	7 YRS	OHA+ISN	NPDR4	NPDR4	0.18	0.26	20	16	0.0215	0.0215	7.1
93	manjula	34/F	8 YRS	OHA	NPDR4	NPDR4	0.26	0.26	16	16	0.0217	0.0216	6.5
94	israel	58/M	2 YRS	OHA	PDR	PDR	0.17	0.19	20	18	0.0216	0.0216	8
95	shanthi	45/F	5 YRS	OHA+ISN	NPDR4	NPDR4	0.19	0.19	18	18	0.0217	0.0217	7.5
96	babu	54/M	3 YRS	OHA	NPDR3	NPDR3	0.21	0.2	18	18	0.0215	0.0215	7.1
97	kathavarayan	39/M	2 YRS	OHA	NO DR	NO DR	0.24	0.26	16	16	0.0216	0.0216	6.01
98	thiruvaruleswari	64/F	14 YRS	OHA+ISN	PDR	PDR	0.19	0.24	18	16	0.0215	0.0214	7.7
99	varadharajan	68/M	7 YRS	OHA	NPDR1	NPDR1	0.26	0.28	14	16	0.0217	0.0217	5.4
100	kathayee	61/F	4 YRS	OHA	NPDR2	NPDR2	0.26	0.26	16	16	0.0216	0.0216	6.4

S .NO	NAME	Age in yrs/ Sex	Duration of DM from Diagnosis	Treatment	Stage of DR		AQ outflow Facility in mlit/min/mm hg		IOP in mm hg		Ocular Rigidity		HbA1c
					RE	LE	RE	LE	RE	LE	RE	LE	
101	lakshmi	42/F	12 YRS	OHA+ISN	NPDR3	NPDR3+CSME	0.28	0.28	14	14	0.0217	0.0217	6.4
102	rajammal	70/F	7 YRS	OHA+ISN	NPDR3+CSME	NPDR3+CSME	0.26	0.26	16	14	0.0216	0.0216	6.5
103	mohan	67/M	15 YRS	OHA	NPDR3+CSME	NPDR3+CSME	0.26	0.26	16	16	0.022	0.022	6.1
104	vasu	40/M	8 YRS	OHA	NPDR2+CSME	NPDR2+CSME	0.26	0.22	16	18	0.022	0.0217	6.6
105	govindraj	52/M	3 YRS	OHA+ISN	PDR	PDR	0.17	0.18	20	20	0.0215	0.0217	8.6
106	vani	35/F	7 YRS	OHA+ISN	NPDR4	NPDR4	0.18	0.2	20	18	0.0214	0.0214	8
107	pankajavalli	40/F	6 YRS	OHA	NO DR	NO DR	0.28	0.28	16	16	0.0216	0.0216	6
108	prema	48/F	4 YRS	OHA	NPDR4	NPDR4	0.24	0.24	16	16	0.0215	0.0215	6.8
109	amirtha kani	61/F	8 YRS	OHA	NPDR1	NPDR1	0.28	0.28	14	14	0.0216	0.0217	5.5
110	govindraj	62/M	12 YRS	OHA	NPDR3	NPDR3	0.26	0.26	16	16	0.0216	0.0216	5.9
111	faridhabanu	60/F	3 YRS	OHA+ISN	PDR	PDR	0.24	0.2	16	18	0.0215	0.0215	8.9
112	kalamani	45/M	15 YRS	OHA	NO DR	NO DR	0.28	0.28	16	16	0.022	0.022	5.9
113	thenmozhi	50/F	7 YRS	OHA	NO DR	NO DR	0.28	0.28	14	14	0.022	0.022	6
114	balamurugan	52/M	8 YRS	OHA+ISN	NPDR4	NPDR4	0.26	0.26	16	16	0.0216	0.0217	6.2
115	srinivasan	49/M	4 YRS	OHA	NPDR3	NPDR3	0.24	0.26	16	16	0.0217	0.0217	6.5
116	raja	67/M	7.3 YRS	OHA+ISN	PDR	PDR	0.2	0.19	18	18	0.0215	0.0215	7.8
117	aswin kumar	42/M	2 YRS	OHA	NO DR	NO DR	0.28	0.26	14	16	0.0217	0.0216	6.25
118	rajeshwari	60/F	7 YRS	OHA	NO DR	NO DR	0.26	0.28	16	14	0.0215	0.0215	5.3
119	sabya	46/F	3 YRS	OHA	NPDR1	NPDR1	0.28	0.28	14	14	0.0216	0.0216	5.1
120	swarnakumar	56/M	9 YRS	OHA+ISN	PDR	PDR	0.19	0.24	18	16	0.0214	0.0214	8.1
121	rajaram	67/M	7 YRS	OHA+ISN	NPDR4	NPDR4	0.24	0.19	16	20	0.0215	0.022	7.7
122	latha	54/F	4 YRS	INSULIN	NPDR3	NPDR3	0.22	0.2	18	18	0.022	0.0215	7.2
123	karpagam	61/F	8 YRS	OHA	NO DR	NO DR	0.26	0.26	16	16	0.0216	0.0217	5
124	sadiq basha	65/M	5 YRS	OHA	NO DR	NO DR	0.24	0.24	18	18	0.0215	0.0215	6.1
125	keerthana	42/F	6 YRS	OHA	NO DR	NO DR	0.24	0.2	18	20	0.022	0.022	6.4
126	vani	57/F	12 YRS	OHA+ISN	PDR	PDR	0.17	0.17	20	22	0.0215	0.0215	8.3
127	pattamal	64/F	8 YRS	OHA	NO DR	NO DR	0.26	0.28	16	16	0.0216	0.0217	5.4
128	krishnan	65/M	12 YRS	OHA+ISN	PDR	PDR	0.24	0.24	16	16	0.0216	0.0216	7.9
129	ramanathan	54/M	10 YRS	OHA	NPDR1	NPDR1	0.22	0.22	18	18	0.0215	0.0215	6.6
130	jayalakshmi	55/F	12 YRS	OHA+ISN	NPDR4	NPDR4	0.2	0.17	18	18	0.0215	0.0215	7.2
131	selvaraj	43/M	3 YRS	OHA	NO DR	NO DR	0.28	0.24	16	18	0.0216	0.0217	5.7
132	menakammal	67/F	4 YRS	OHA	NO DR	NO DR	0.28	0.28	16	16	0.0215	0.0215	5.4
133	pachiammal	57/F	9 YRS	INSULIN	PDR	PDR	0.17	0.19	20	18	0.022	0.022	7.1
134	jagadeesh	45/M	5 YRS	OHA	NO DR	NO DR	0.28	0.26	16	16	0.0214	0.0214	5.9

S .NO	NAME	Age in yrs/ Sex	Duration of DM from Diagnosis	Treatment	Stage of DR		AQ outflow Facility in mlit/min/mm hg		IOP in mm hg		Ocular Rigidity		HbA1c
					RE	LE	RE	LE	RE	LE	RE	LE	
135	munuswamy	57/M	9 YRS	OHA+ISN	NPDR4	NPDR4	0.2	0.17	18	16	0.0215	0.0215	7.4
136	dilliammal	45/F	7.3 YRS	OHA	NO DR	NO DR	0.22	0.24	18	16	0.0216	0.0216	6.1
137	srinivasan	55/M	10 YRS	OHA+ISN	NPDR4	NPDR4	0.18	0.18	16	18	0.0217	0.0217	7.6
138	kamala	61/F	12 YRS	OHA	NO DR	NO DR	0.24	0.24	16	16	0.0215	0.0215	5.7
139	gopal rao	54/M	2 YRS	OHA	NO DR	NO DR	0.26	0.26	16	16	0.0216	0.0216	5.4
140	baskar	56/M	7 YRS	OHA+ISN	PDR	PDR	0.17	0.24	20	16	0.0215	0.0215	6.9
141	kesavan	67/M	7 YRS	OHA	NPDR4	NPDR4	0.26	0.26	16	16	0.0217	0.0217	6.6
142	moorthy	62/M	5 YRS	OHA	NO DR	NO DR	0.22	0.2	20	20	0.022	0.022	6.9
143	palani	56/M	8 YRS	INSULIN	PDR	PDR	0.24	0.28	16	14	0.0215	0.0215	6.7
144	kandasamy	49/M	5 YRS	OHA	NO DR	NO DR	0.24	0.26	16	16	0.0214	0.0214	6.5
145	kalimuthu	58/M	12 YRS	OHA	NO DR	NO DR	0.28	0.28	14	14	0.0216	0.0216	6.1
146	kannamal	68/M	11.7 YRS	OHA+ISN	NPDR4	NPDR4	0.22	0.24	18	16	0.0215	0.0215	7.6
147	manikam	63/M	9 YRS	OHA	NO DR	NO DR	0.28	0.28	14	14	0.0217	0.0216	6.2
148	nagamma	54/F	8 YRS	OHA+ISN	PDR	PDR	0.2	0.17	18	20	0.0215	0.0215	8.2
149	kumutha	63/F	11 YRS	OHA	NO DR	NO DR	0.28	0.28	14	14	0.0214	0.0214	5.4
150	mathan kumar	55/M	8 YRS	OHA+ISN	PDR	PDR	0.24	0.19	16	18	0.0216	0.0216	7.5

FOLLOW-UP CHART

S.NO.	NAME	HbA1C			FUNDUS						AQ Outflow facility mlit/min/mmhg						IOP in mm hg						GLAUCOMATOUS OPTIC NERVE HEAD CHANGES						GLAUCOMATOUS FIELD DEFECTS IN PERIMETRY					
		V1	V2	V3	RE			LE			RE			LE			RE			LE			RE			LE			RE			LE		
					V1	V2	V3	V1	V2	V3	V1	V2	V3	V1	V2	V3	V1	V2	V3	V1	V2	V3	V1	V2	V3	V1	V2	V3	V1	V2	V3	V1	V2	V3
1	shanmugam	7.1	7	6.4	NPDR1	NPDR1	NPDR1	NPDR1	NPDR1	NPDR1	0.19	0.21	0.2	0.2	0.22	0.22	20	18	18	20	18	18	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
2	suseela	6.7	6.7	6.5	NPDR2	NPDR2	NPDR2	NPDR2	NPDR2	NPDR2	0.19	0.22	0.22	0.2	0.22	0.22	20	18	18	18	18	16	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
3	dhanalakshmi	7.4	7.9	7.5	NPDR3	NPDR3	NPDR3	NPDR3	NPDR3	NPDR3	0.2	0.19	0.19	0.19	0.18	0.18	18	20	20	20	20	22	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
4	kowsalya	7.2	7.3	7.1	NPDR3	NPDR3	NPDR3	NPDR3	NPDR3	NPDR3	0.19	0.2	0.2	0.19	0.19	0.2	20	18	18	20	20	20	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
5	beenaliraj	6.9	7.1	LFU	NPDR3	NPDR3	LFU	NPDR3	NPDR3	LFU	0.2	0.2	LFU	0.19	0.2	LFU	18	18	LFU	18	18	LFU	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
6	noorjahan	7.1	7.1	7	NPDR3	NPDR3	NPDR3	NPDR3	NPDR3	NPDR3	0.19	0.19	0.2	0.19	0.19	0.2	18	18	20	18	18	20	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
7	shanti	7.3	7.3	7.1	NPDR3	NPDR3	NPDR3	NPDR3	NPDR3	NPDR3	0.19	0.19	0.19	0.19	0.19	0.19	20	20	18	20	18	20	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
8	lakshmi	6.6	6.8	LFU	NPDR3	NPDR3	LFU	NPDR3	NPDR3	LFU	0.19	0.19	LFU	0.26	0.24	LFU	18	18	LFU	16	18	LFU	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
9	dhanalakshmi	7.3	7.3	7	NPDR4	NPDR4	NPDR4	NPDR4	NPDR4	NPDR4	0.19	0.19	0.2	0.18	0.19	0.19	18	20	18	20	20	18	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
10	meena	7.3	7.4	LFU	NPDR4	NPDR4	LFU	NPDR4	NPDR4	LFU	0.18	0.17	LFU	0.18	0.17	LFU	18	18	LFU	18	18	LFU	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
11	vellathai	7.1	7	7	NPDR4	NPDR4	NPDR4	NPDR4	NPDR4	NPDR4	0.18	0.19	0.19	0.17	0.19	0.19	20	18	18	18	18	18	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
12	vishalatchi	7.1	7.2	LFU	NPDR4	NPDR4	LFU	NPDR4	NPDR4	LFU	0.2	0.19	LFU	0.18	0.18	LFU	18	18	LFU	18	18	LFU	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
13	parvathy	7.5	7.9	7.5	NPDR4	NPDR4	NPDR4	NPDR4	NPDR4	NPDR4	0.18	0.17	0.18	0.17	0.17	0.19	18	20	20	20	20	20	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
14	padmavathy	7.1	7.4	7.5	NPDR4	NPDR4	NPDR4	NPDR4	NPDR4	NPDR4	0.18	0.18	0.17	0.26	0.22	0.22	20	20	20	16	18	18	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
15	mariappa	7.1	7.4	7.4	NPDR4	NPDR4	NPDR4	NPDR4	NPDR4	NPDR4	0.19	0.18	0.18	0.18	0.18	0.17	16	18	18	18	20	20	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
16	sundar	7.3	7.7	7.9	NPDR4	NPDR4	NPDR4	NPDR4	NPDR4	NPDR4	0.18	0.17	0.17	0.2	0.17	0.17	18	22	22	18	22	24	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	YES
17	anushya	6.9	LFU	LFU	NPDR4	LFU	LFU	NPDR4	LFU	LFU	0.24	LFU	LFU	0.19	LFU	LFU	16	LFU	LFU	18	LFU	LFU	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
18	shanthi	7.5	7.3	LFU	NPDR4	NPDR4	LFU	NPDR4	NPDR4	LFU	0.19	0.18	LFU	0.19	0.18	LFU	18	18	LFU	18	18	LFU	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
19	vani	8	8	8.3	NPDR4	NPDR4	NPDR4	NPDR4	NPDR4	NPDR4	0.18	0.16	0.16	0.2	0.18	0.17	20	24	24	18	20	24	NO	NO	YES	NO	NO	NO	NO	NO	YES	NO	NO	NO
20	rajaram	7.7	7.5	7.7	NPDR4	NPDR4	NPDR4	NPDR4	NPDR4	NPDR4	0.24	0.22	0.22	0.19	0.19	0.18	16	16	18	20	22	22	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
21	jayalakshmi	7.2	7.4	LFU	NPDR4	NPDR4	LFU	NPDR4	NPDR4	LFU	0.2	0.18	LFU	0.17	0.17	LFU	18	18	LFU	18	20	LFU	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
22	munusamy	7.4	7.2	LFU	NPDR4	NPDR4	LFU	NPDR4	NPDR4	LFU	0.2	0.2	LFU	0.17	0.17	LFU	18	18	LFU	16	20	LFU	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
23	srinivasan	7.6	7.4	7.4	NPDR4	NPDR4	NPDR4	NPDR4	NPDR4	NPDR4	0.18	0.18	0.18	0.18	0.18	0.18	16	16	16	18	18	18	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
24	gunasekar	8.1	7.8	7.8	PDR	PDR	PDR	PDR	PDR	PDR	0.17	0.17	0.18	0.19	0.18	0.18	20	20	20	18	18	18	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
25	rajaram	7.4	7.4	LFU	PDR	PDR	LFU	PDR	PDR	LFU	0.19	0.18	LFU	0.19	0.19	LFU	18	18	LFU	18	18	LFU	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
26	kandasamy	8	8	7.9	PDR	PDR	PDR	PDR	PDR	PDR	0.22	0.22	0.22	0.17	0.18	0.18	16	16	18	18	18	18	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
27	senthil	7.9	LFU	LFU	PDR	LFU	LFU	PDR	LFU	LFU	0.18	LFU	LFU	0.22	LFU	LFU	18	LFU	LFU	16	LFU	LFU	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
28	devika	8.3	8.3	8.1	PDR	PDR	PDR	PDR	PDR	PDR	0.19	0.19	0.19	0.19	0.19	0.17	18	18	18	18	20	20	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
29	gunasekar	7.9	7.7	LFU	PDR	PDR	LFU	PDR	PDR	LFU	0.17	0.17	LFU	0.18	0.18	LFU	20	20	LFU	18	18	LFU	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
30	poochendu	7.9	7.9	8.1	PDR	PDR	PDR	PDR	PDR	PDR	0.18	0.18	0.17	0.18	0.17	0.17	18	18	20	18	20	20	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
31	rani	8.4	8.1	8.1	PDR	PDR	PDR	PDR	PDR	PDR	0.17	0.16	0.17	0.17	0.17	0.17	20	20	22	20	20	20	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
32	vanishree	7.6	7.7	LFU	PDR	PDR	LFU	PDR	PDR	LFU	0.19	0.18	LFU	0.17	0.17	LFU	18	18	LFU	20	20	LFU	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
33	thiruvaruleswari	7.7	LFU	LFU	PDR	LFU	LFU	PDR	LFU	LFU	0.19	LFU	LFU	0.24	LFU	LFU	18	LFU	LFU	16	LFU	LFU	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
34	faridhabanu	8.9	8.3	8.5	PDR	PDR	PDR	PDR	PDR	PDR	0.24	0.21	0.2	0.2	0.2	0.18	16	18	18	18	20	20	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
35	dhamayanthi	8.5	8.2	8.4	PDR	PDR	PDR	PDR	PDR	PDR	0.24	0.21	0.21	0.2	0.2	0.18	16	18	18	18	18	20	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
36	govindraj	8.6	8.4	LFU	PDR	PDR	LFU	PDR	PDR	LFU	0.17	0.17	LFU	0.18	0.17	LFU	20	20	LFU	20	20	LFU	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
37	raja	7.8	7.4	LFU	PDR	PDR	LFU	PDR	PDR	LFU	0.2	0.2	LFU	0.19	0.19	LFU	18	18	LFU	18	18	LFU	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
38	swarnakumar	8.1	7.7	LFU	PDR	PDR	LFU	PDR	PDR	LFU	0.19	0.19	LFU	0.24	0.24	LFU	18	18	LFU	16	16	LFU	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
39	vani	8.3	8.3	LFU	PDR	PDR	LFU	PDR	PDR	LFU	0.17	0.17	LFU	0.17	0.17	LFU	20	22	LFU	22	22	LFU	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
40	pachiammal	7.1	LFU	LFU	PDR	LFU	LFU	PDR	LFU	LFU	0.17	LFU	LFU	0.19	LFU	LFU	20	LFU	LFU	18	LFU	LFU	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
41	baskar	6.9	7.1	LFU	PDR	PDR	LFU	PDR	PDR	LFU	0.17	0.17	LFU	0.24	0.17	LFU	20	20	LFU	16	16	LFU	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
42	nagamma	8.2	8.3	LFU	PDR	PDR	LFU	PDR	PDR	LFU	0.2	0.19	LFU	0.17	0.17	LFU	18	18	LFU	20	20	LFU	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
43	mathankumar	7.5	LFU	LFU	PDR	LFU	LFU	PDR	LFU	LFU	0.24	LFU	LFU	0.19	LFU	LFU	16	LFU	LFU	18	LFU	LFU	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO

Direct determination of facility of aqueous outflow from schiotz scale reading(5.5 GM WT).

(Courtesy Committee On Tonometry, American Academy Of Ophthalmology,1965)

Initial reading	IOP Mm H g	Change from initial to final scale									
		0.50	1.00	1.50	2.00	2.50	3.00	3.50	4.00	4.50	5.00
0.50 0.75	38 36	0.06 0.06	0.14 0.13	0.24 0.22	0.39 0.35	0.61 0.53	0.94 0.78	_	_	_	_
1.00 1.25	35 33	0.05 0.05	0.12 0.12	0.21 0.19	0.32 0.29	0.47 0.42	0.66 0.59	0.94 0.81	_	_	_
1.50 1.75	32 30	0.05 0.05	0.11 0.11	0.18 0.17	0.27 0.25	0.38 0.35	0.53 0.48	0.71 0.64	0.94 0.83	_	_
2.00 2.25	29 28	0.05 0.05	0.10 0.10	0.16 0.16	0.24 0.23	0.33 0.31	0.44 0.41	0.58 0.53	0.74 0.68	0.93 0.84	_
2.50 2.75	27 25	0.04 0.04	0.09 0.09	0.15 0.15	0.22 0.21	0.30 0.28	0.39 0.37	0.49 0.46	0.62 0.58	0.76 0.70	0.92 0.84
3.00 3.25	24 23	0.04 0.04	0.09 0.09	0.14 0.14	0.20 0.20	0.27 0.26	0.35 0.33	0.44 0.42	0.54 0.51	0.65 0.62	0.78 0.74
3.50 3.75	22 22	0.04 0.04	0.08 0.08	0.13 0.13	0.19 0.19	0.25 0.25	0.32 0.31	0.40 0.38	0.49 0.47	0.59 0.56	0.70 0.66
4.00 4.25	21 20	0.04 0.04	0.08 0.08	0.13 0.13	0.18 0.18	0.24 0.24	0.30 0.30	0.37 0.36	0.45 0.43	0.54 0.52	0.63 0.60
4.50 4.75	19 18	0.04 0.04	0.08 0.08	0.12 0.12	0.17 0.17	0.23 0.23	0.29 0.28	0.35 0.34	0.42 0.41	0.50 0.48	0.58 0.56
5.00 5.25	17 17	0.04 0.04	0.08 0.08	0.12 0.12	0.17 0.17	0.22 0.22	0.27 0.27	0.33 0.33	0.40 0.39	0.47 0.46	0.54 0.53
5.50 5.75	16 15	0.04 0.04	0.08 0.08	0.12 0.12	0.16 0.16	0.21 0.21	0.26 0.26	0.32 0.32	0.39 0.38	0.45 0.44	0.52 0.50
6.00 6.25	15 14	0.03 0.03	0.07 0.07	0.11 0.11	0.15 0.15	0.20 0.20	0.25 0.25	0.31 0.31	0.37 0.37	0.43 0.43	0.49 0.49
6.50 6.75	13 13	0.03 0.03	0.07 0.07	0.11 0.11	0.15 0.15	0.20 0.20	0.25 0.24	0.30 0.30	0.36 0.36	0.42 0.41	0.48 0.47
7.00 7.50	12 11	0.03 0.03	0.07 0.07	0.11 0.11	0.15 0.15	0.20 0.19	0.24 0.24	0.29 0.29	0.35 0.34	0.40 0.39	0.46 0.45
8.00 8.50	10 09	0.03 0.03	0.07 0.07	0.11 0.11	0.15 0.15	0.19 0.19	0.24 0.23	0.29 0.28	0.34 0.33	0.39 0.39	0.45 0.45
9.00 9.50	08 08	_	_	0.11 0.11	0.15 0.15	0.19 0.19	0.23 0.23	0.28 0.28	0.33	_	_
10.00	07	_	_	0.11	0.15	0.19	0.23		_	_	_

Direct determination of facility of aqueous outflow from schiotz scale reading(7.5 GM WT).

(Courtesy Committee On Tonometry, American Academy Of Ophthalmology,1965)

Initial reading	IOP Mm H g	Change from initial to final scale									
		0.50	1.00	1.50	2.00	2.50	3.00	3.50	4.00	4.50	5.00
0.50 0.75	54 52	0.05 0.05	0.12 0.12	0.23 0.21	0.41 0.36	0.76 0.63	_	_	_	_	_
1.00 1.25	50 48	0.04 0.04	0.11 0.10	0.19 0.17	0.32 0.29	0.50 0.44	0.79 0.65	_	_	_	_
1.50 1.75	46 44	0.04 0.04	0.09 0.09	0.16 0.15	0.26 0.24	0.30 0.34	0.35 0.48	0.79 0.67	_	_	_
2.00 2.25	42 41	0.04 0.04	0.09 0.09	0.14 0.14	0.22 0.20	0.31 0.29	0.43 0.39	0.58 0.52	0.78 0.68	_	_
2.50 2.75	39 37	0.04 0.04	0.08 0.08	0.13 0.13	0.19 0.18	0.27 0.25	0.36 0.33	0.47 0.43	0.60 0.54	0.77 0.68	_
3.00 3.25	36 34	0.03 0.03	0.07 0.07	0.12 0.12	0.17 0.17	0.23 0.22	0.31 0.29	0.40 0.37	0.50 0.46	0.62 0.57	0.76 0.69
3.50 3.75	33 31	0.03 0.03	0.07 0.07	0.11 0.11	0.16 0.16	0.21 0.21	0.28 0.26	0.35 0.33	0.43 0.41	0.53 0.49	0.63 0.59
4.00 4.25	30 29	0.03 0.03	0.06 0.06	0.10 0.10	0.15 0.15	0.20 0.19	0.25 0.25	0.32 0.30	0.39 0.37	0.46 0.44	0.55 0.52
4.50 4.75	29 27	0.03 0.03	0.06 0.06	0.10 0.10	0.14 0.14	0.18 0.18	0.24 0.23	0.29 0.28	0.35 0.34	0.42 0.40	0.50 0.47
5.00 5.25	26 25	0.03 0.03	0.06 0.06	0.10 0.10	0.13 0.13	0.17 0.17	0.22 0.22	0.27 0.27	0.33 0.32	0.39 0.38	0.45 0.43
5.50 5.75	24 23	0.03 0.03	0.06 0.06	0.09 0.09	0.13 0.13	0.16 0.16	0.21 0.21	0.26 0.26	0.31 0.31	0.37 0.36	0.42 0.41
6.00 6.25	22 21	0.03 0.03	0.06 0.06	0.09 0.09	0.12 0.12	0.16 0.16	0.20 0.20	0.25 0.25	0.30 0.29	0.35 0.34	0.40 0.39
6.50 6.75	20 19	0.03 0.03	0.05 0.05	0.09 0.09	0.12 0.12	0.15 0.15	0.18 0.18	0.24 0.24	0.28 0.28	0.33 0.33	0.38 0.38
7.00 7.50	18 17	0.03 0.03	0.05 0.05	0.08 0.08	0.12 0.12	0.15 0.15	0.19 0.19	0.24 0.23	0.27 0.27	0.32 0.31	0.37 0.36
8.00 8.50	16 14	0.03 0.03	0.05 0.05	0.08 0.08	0.11 0.11	0.15 0.15	0.18 0.18	0.22 0.22	0.26 0.26	0.30 0.30	0.35
9.00 9.50	13 12	0.03 0.03	0.05 0.05	0.08 0.08	0.11 0.11	0.15 0.15	0.18 0.18	0.22 0.22	0.25	_	_
10.00	11	0.03	0.05	0.08	0.11	0.14	0.18	_	_	_	_